Important definitions

Microbiology is the study of living organisms of **microscopic size**

Eukaryotic cells

Have a true nucleus

- **Multiple** chromosomes
- Nuclear membrane

Ex.:Algae, Protozoa & Fungi

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Prokaryotic cells

Have a **primitive** nucleus

- **Single** chromosome
- **No** nuclear membrane

Ex.: Bacteria

Viruses

- **The smallest infectious org.**
- Aren't true cells

Obligate IC organism

Rely on host cells for replication

Items of general bacteriology

1-STRUCTURE OF BACTERIAL CELLS.

2-ANTIMICROBIALS.

3-BACTERIAL GENETICS.

4- HOST MICROBE RELATIONSHIP.

5-BACTERIAL GROWTH & CLASSIFICATION

Bacterial Cell Structure

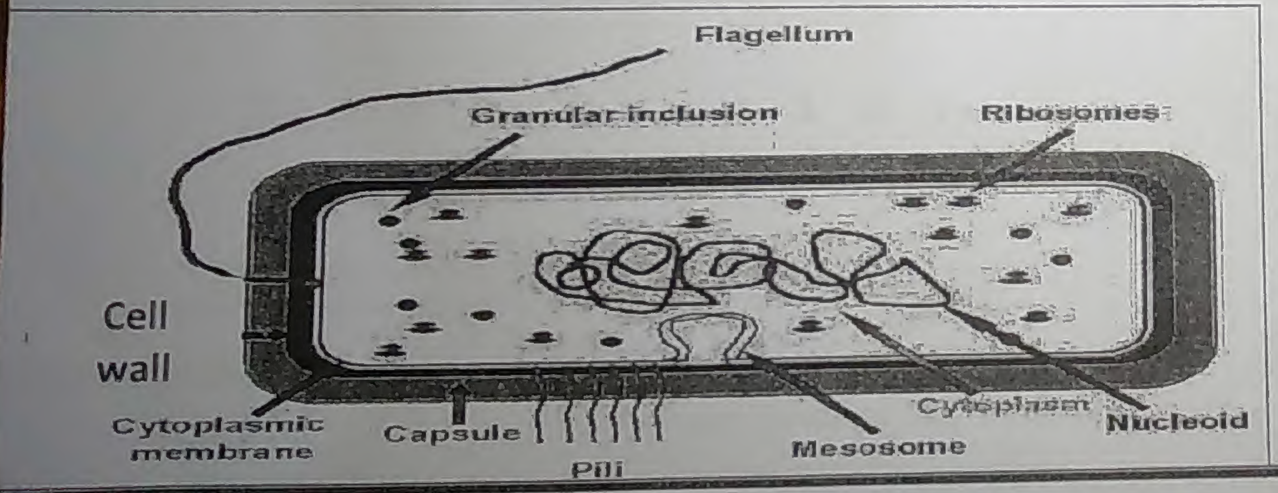
A - Cell wall (B1) : Protection

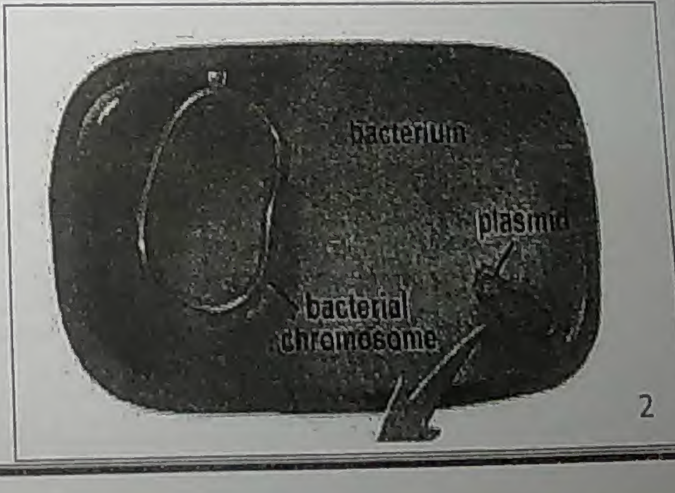
B - Structures inside cell wall

- **1-Cell membrane (B2)**
  - Selective permeability
- **2- Cytoplasm**
  - **i.Ribosomes (B3)**
    - Protein synthesis
  - **ii.Inclusion granules**
    - Storage
  - **iii.Bacterial genome**
    - **Nucleoid (B4)**
      - **Single** circular Ds DNA
    - **Plasmid**
      - Circular Ds DNA
      - **Extra**chromosomal
      - < chromosome

C - Structures outside cell wall

- **1-Capsule** — Protection
- **2-Flagella** — Motility
- **3-Pili** — Attachment
- **4-Spores** — Protection





Cytoplasmic membrane (CM)

A-Structure

Phospholipid bilayer
containing proteins
(as eukaryotic CM)

No cholesterol
except in
Mycoplasma
الشريك المخالف

Mesosomes

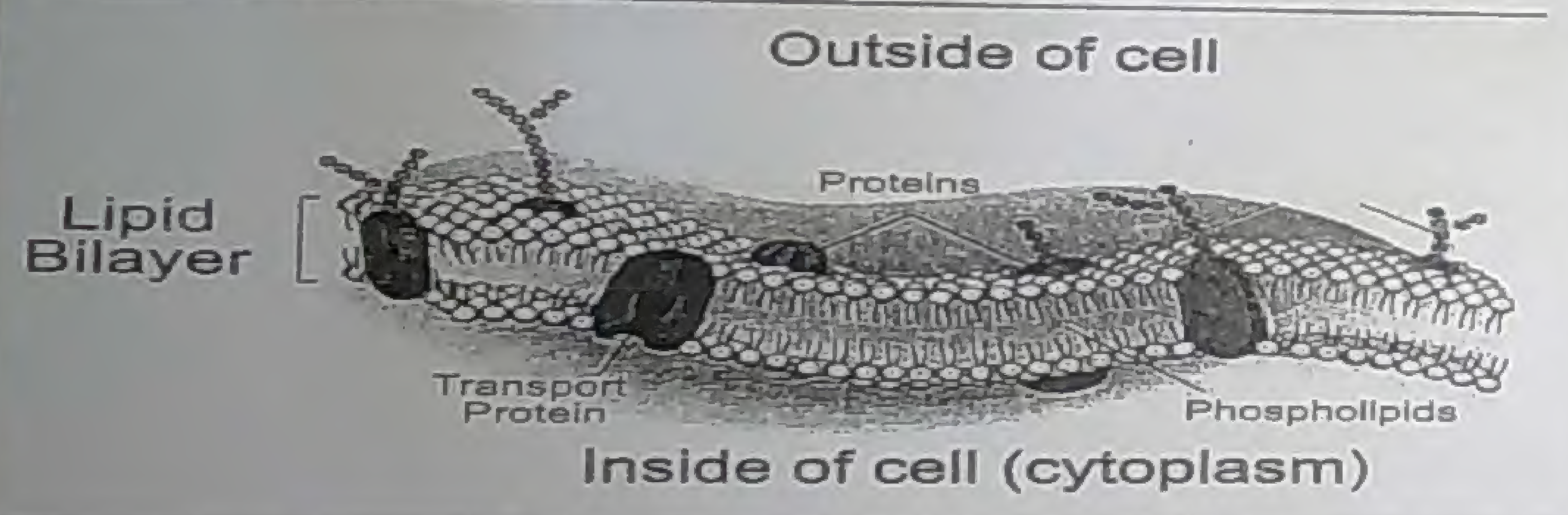
SERK
Meso
cholesterol

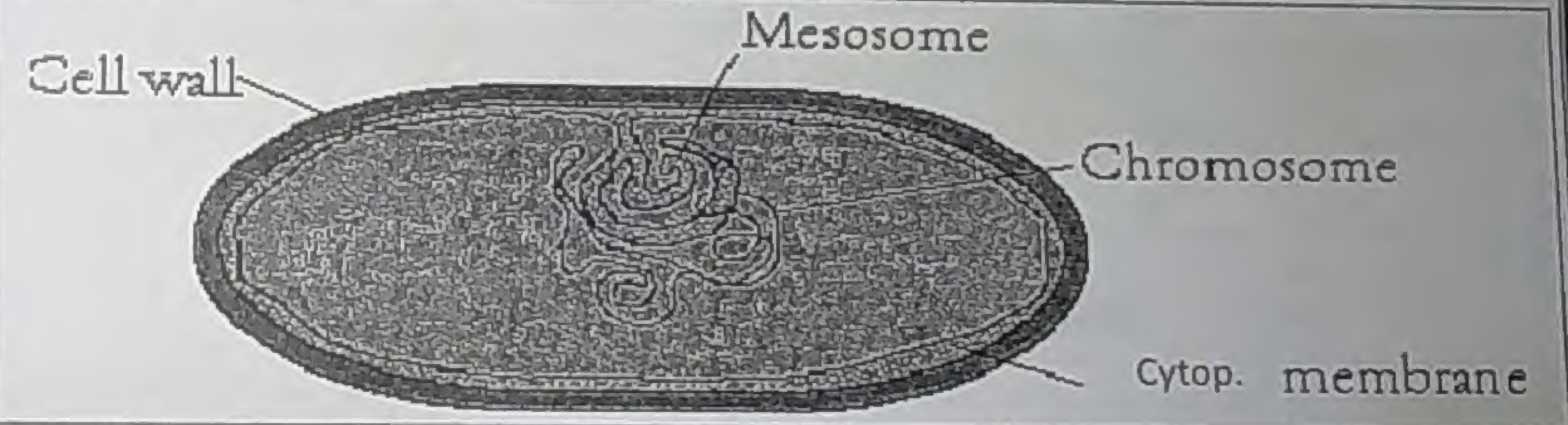
Structure
Invagination of CM
inside cytoplasm

Types
i. Lateral
ii. Septal

Function
Cell division
SM are the site of
chromosomal attachement

Structure of the Cell Membrane





B-Functions

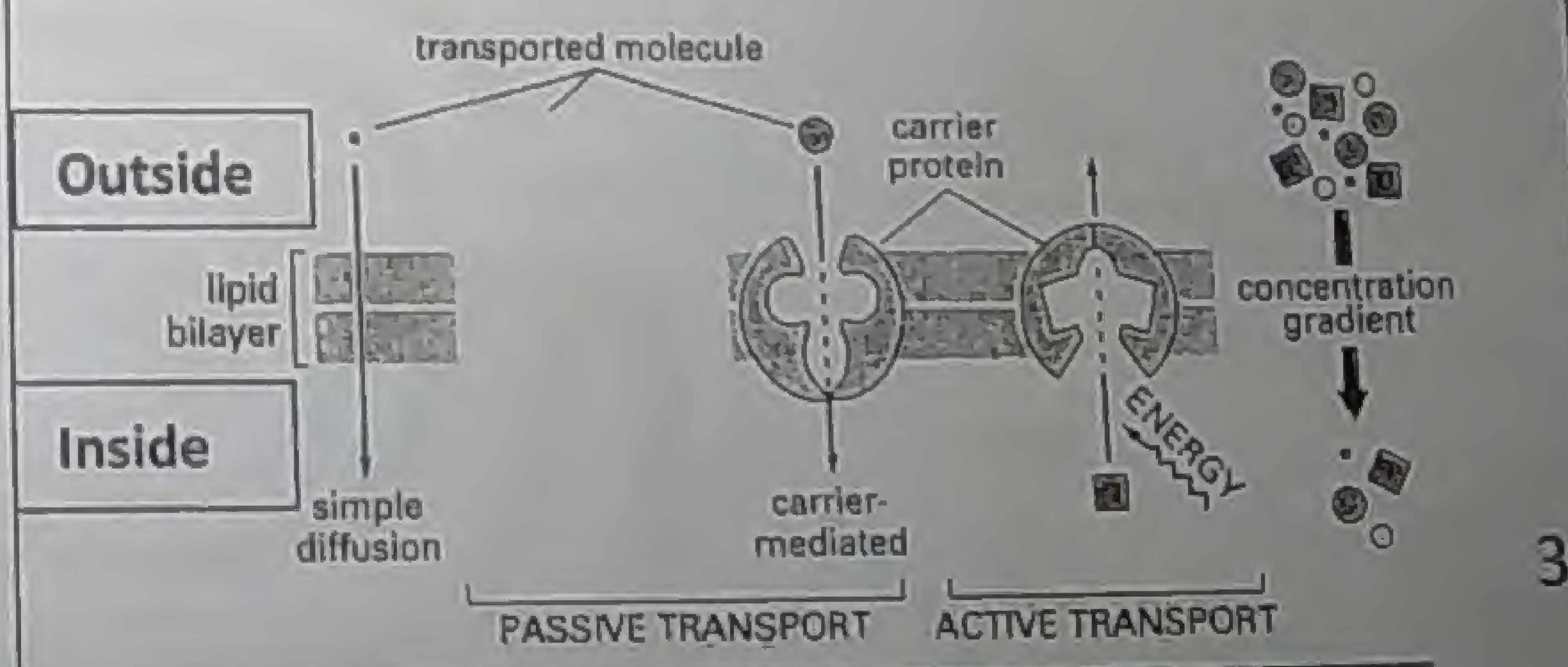
1 - Selective transport

SERC

Molecules move across the membrane by

Simple diffusion

Active transport
Against conc.gradient → Requires energy



2- Excretion of extracellular

Enzymes — Exotoxins

Destructive

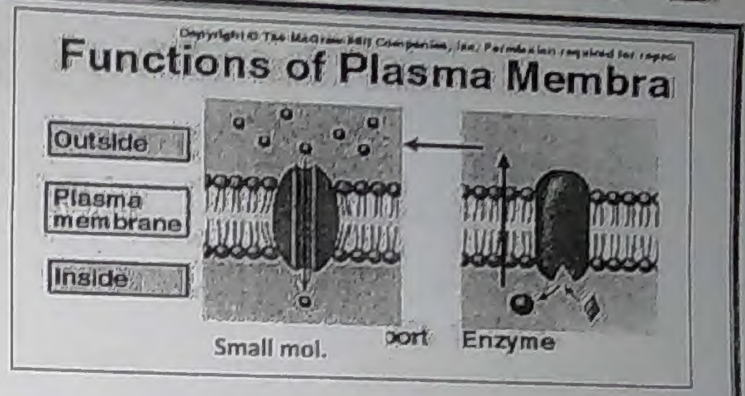
Degrade **antibiotics**

Hydrolytic

Digest large food mol.

Subunits **small enough**

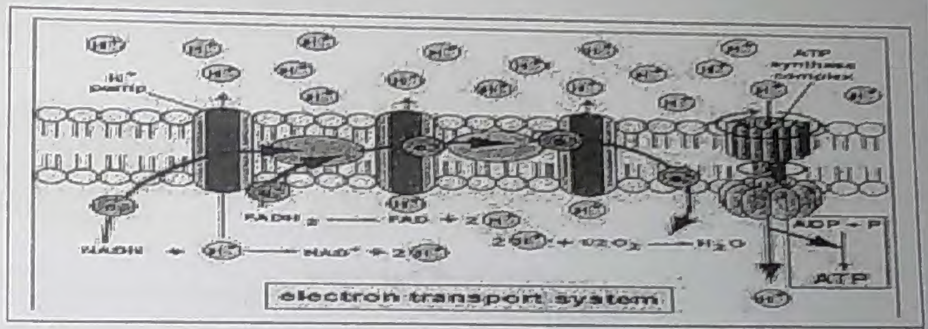
to penetrate the cell



3 - Respiration & generation of ATP

Contain **respiratory & cytochrome enzymes**

(as ***mitochondrial*** membrane in eukaryotes)



4 - Chemotactic systems

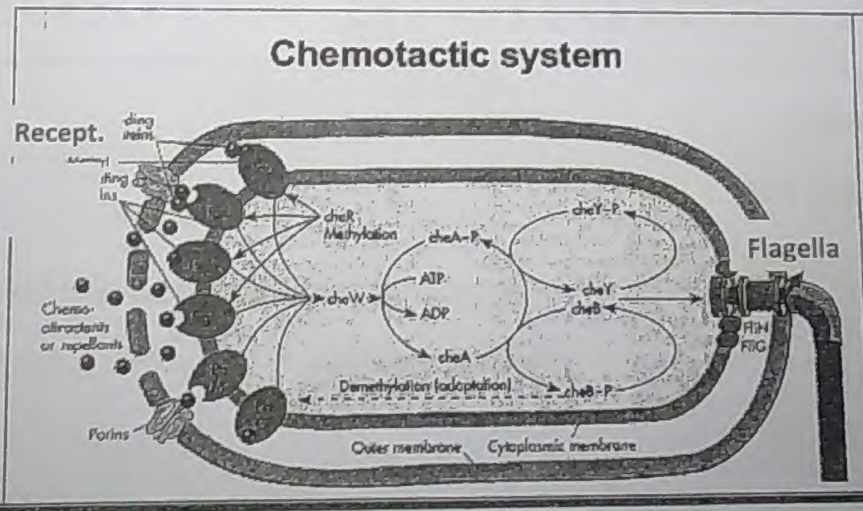
Expresses specific **receptors**

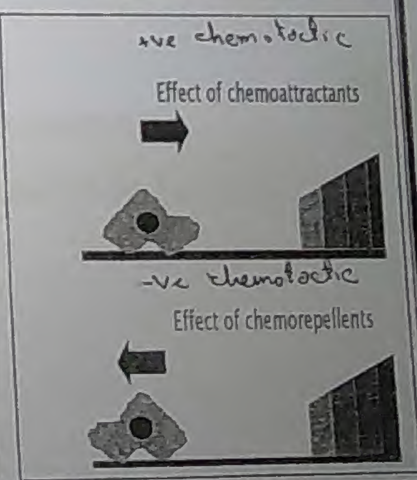
⬇

Bind attractants & repellants

⬇

Send signals to cell interior





Cytoplasm

Ribosomes

Structure

Ribosomal RNA + proteins

Subunits

Large **50S**

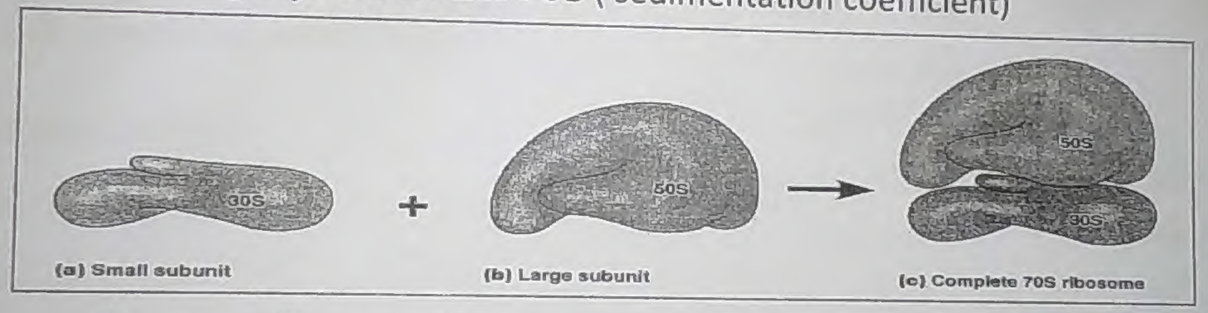
Small **30S**

Aggregate during protein synthesis

polyribosome: 70S (sedimentation coefficient)

Functions

Protein synthesis



Inclusion granules

Types

Nutrient reserve

for cell metabolism

Stored energy

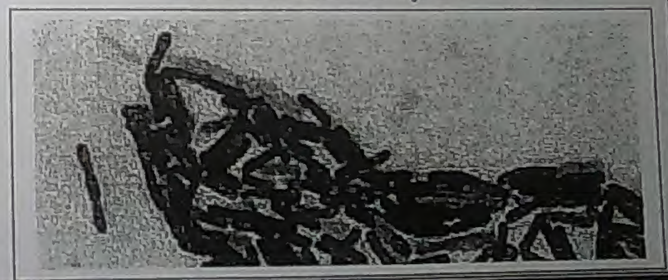
e.g ***Volutin granules*** in Diphtheria

Polyphosphate

Metachromatic

Characters

Not essential or permanent



Cell Wall

I-Structure (absent in Mycoplasma)

Differs between **Gram + ve** & **Gram -ve** bacteria

A - Peptidoglycan

Q Give Reason: cell wall of gram+ve bacteria is stronger than gram-ve bacteria?

G+ve bacteria	G-ve bacteria
1.Thick : 40 sheets → ***50%*** of CW thickness → ***Stronger*** CW	***1.Thin*** : 1-2 sheets → ***5-10%*** of CW → ***Weaker*** CW

2.Each sheet is formed of **alternating**

N-actetyl muramic acid & N-acetyl glucoseamine.

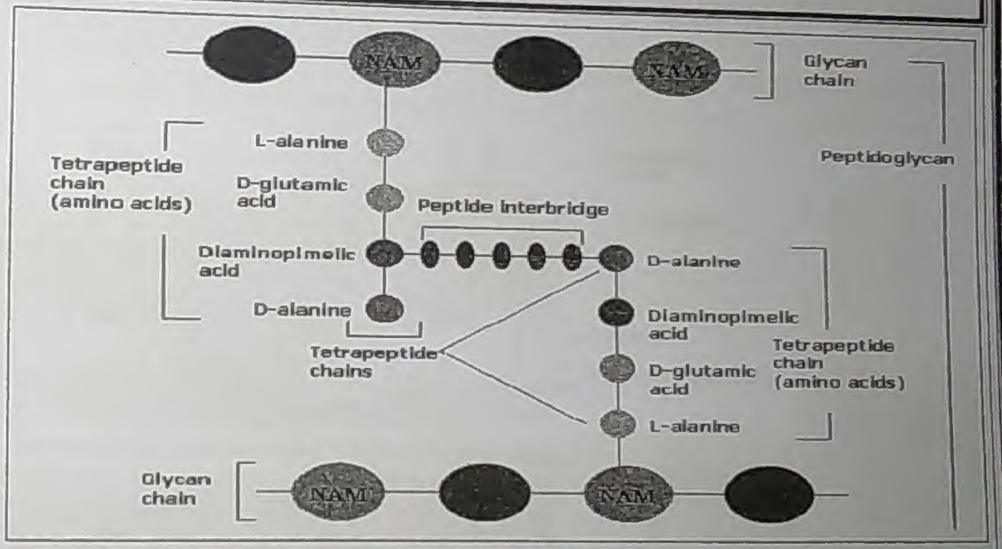
3.Sheets are connected by :

i . **4 a.a. (tetrapeptide) side chains** (attached to NAM)

ii.Identical cross linking peptide bridges

F : ***Rigidity (osmotic barrier)*** **(1)**

Supports weak CM → prevents osmotic rupture

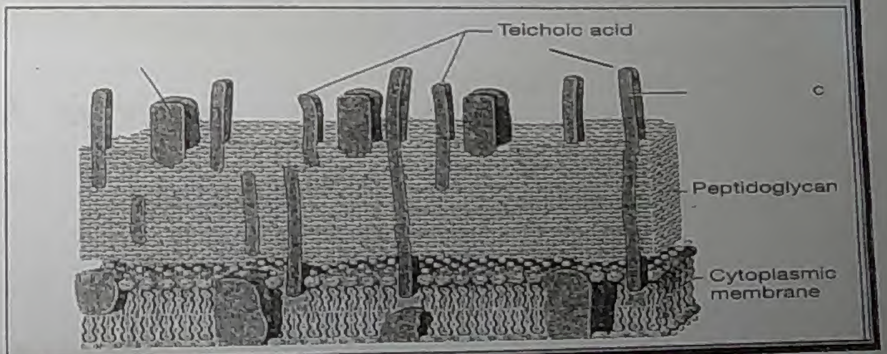


B - Other layers of G+ve bacteria

Techoic acid (found also in CM)

S : Glycerol or ribitol phosphate

F : Major Ag (2.i)



C – Other layers of G - ve bacteria

1. Outer membrane (OM)

a-Inner part

Phospholipid bilayer

similar

in composition

to CM

b-Outer part : Lipopolysaccharides (LPS)

i.Inner

Lipid A

↓

Endotoxin(3)

(Virulence F)

Released only after Bacterial death.

ii.Middle

Polysaccharide

core

iii.Outer

Polysaccharide side chains

↓

Somatic O Ag

(2.ii)

c-Porins proteins

Special channels

in OM

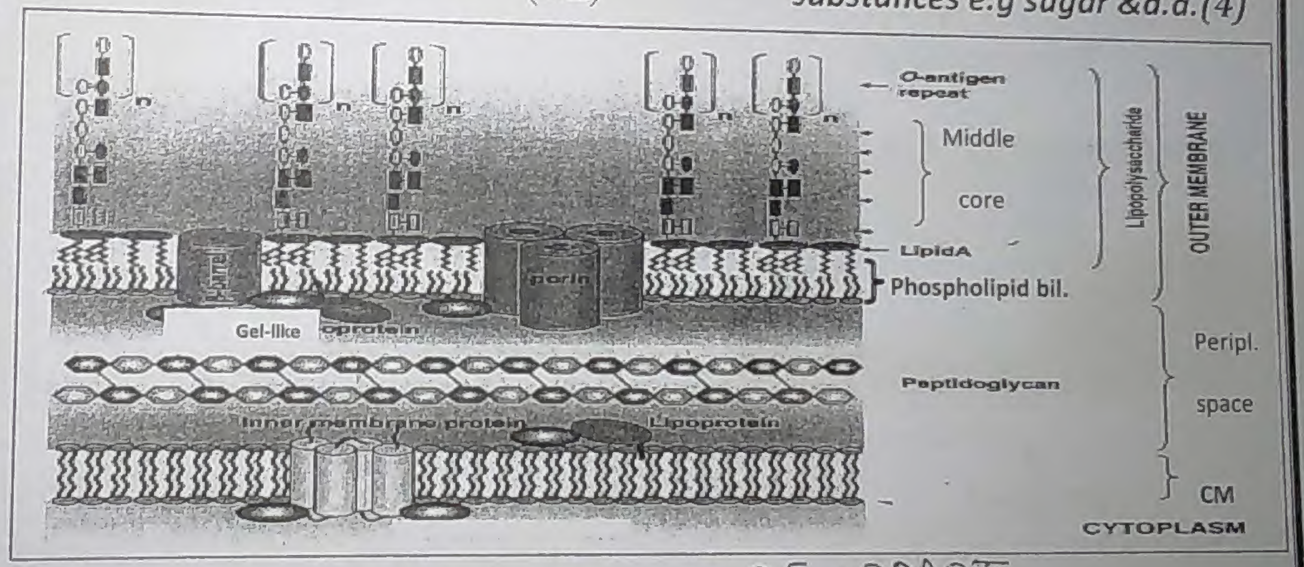
↓

Passive diffusion of low MW substances e.g sugar &a.a.(4)

2. Periplasmic space

Between CM & OM :

contain **PG + gel-like protein**



II – Functions 2S + DOAPT

Staining reaction	*Shape of bacteria*	*Cell division*	*Osmotic barrier (1) &Antigenic (2. i &ii)*
	Cocci,bacilli,spirilla.		*Toxic (3) & Passive diffusion (4)*

Structures outside cell wall

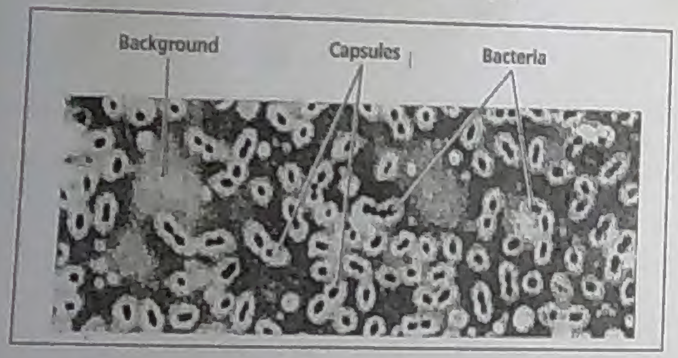
A- Capsule

1-Site

- **Outermost**
- Only in **some bacteria**
- Usually **in vivo**

2-Structure

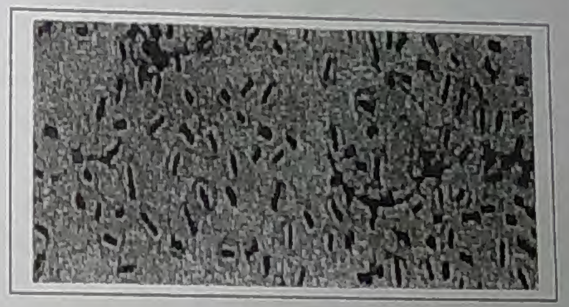
- **Polysaccharide**(usually)
- Polypeptide
- Hyaluronic acid



3-Stain

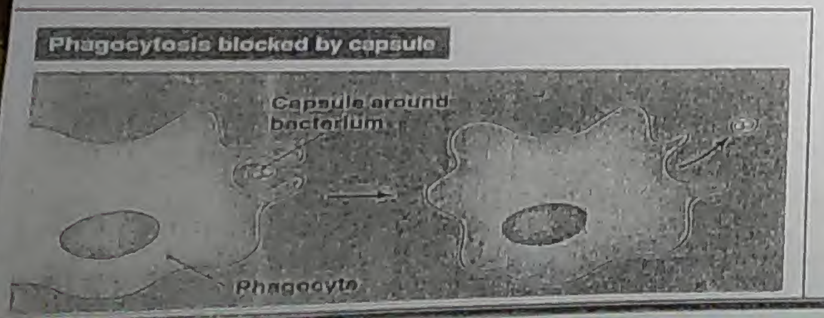
Capsular stain (not stained by Gram)

?!



4 -Functions AAA

- ***Antiphagocytic*** → VF
- **Attachment** to mucus membrane
- **Antigenic**



virulence [VF]

stimulate the immune respone in the body of host

B - Appendages

Flagella

A-Structure & shape

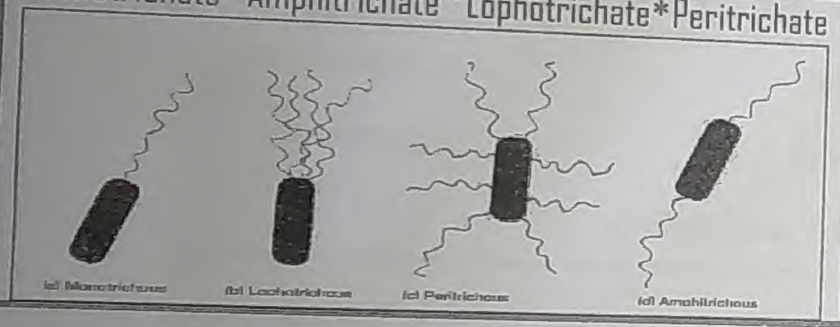
i.Formed of a protein cd : **flagellin**

ii.Long & thick

iii.Arise from cytoplasm & extrudes through CW

B-Arrangement

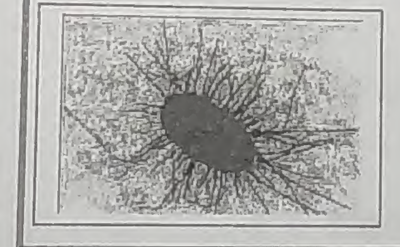
*Monotrichate *Amphitrichate *Lophotrichate*Peritrichate



C-Types & functions

1-Motility

2-Antigenic : H Ag



Fimbria (pili)

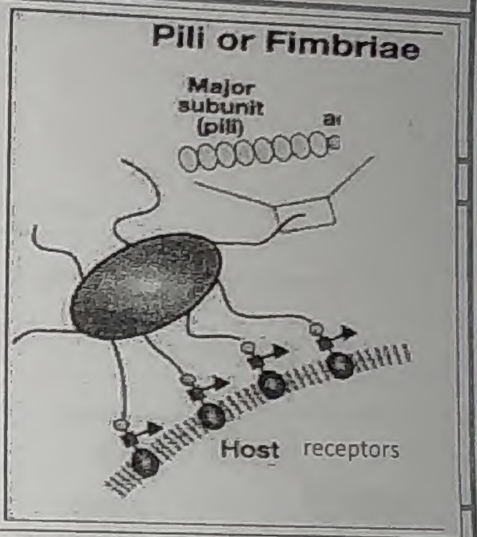
A-Structure & shape

i.Formed of a protein cd : **pilin**

ii.Short & thin

B-Arrangement

Peritrichate



C-Types & functions

1-Ordinary pili

Adherence → *VF* *Antigenic*

the attachment resists the mucus secretion.

Attachement of bacteria

to specific recept.on human cells

2-Sex pili : Conjugation

Gene transfer between bacteria

D-Stain

?) ***Flagellar stain*** (not stained by Gram)

C - Endospores

A-Definition

Highly *resistant resting* phase formed by **Bacillus** & **Clostridium** for protection

B-Sporulation

1-Triggering &Site

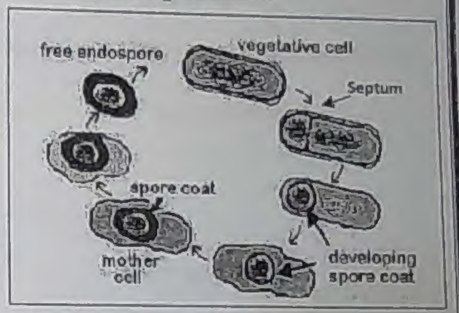
In vitro : by onset of unfavorable environmental conditions

- ***Depletion of nutrients***
- Accumulation of ***toxic metabolites***
- Changes in ***growth requirements*** e.g Moisture , temperature

2-Stages (structure)

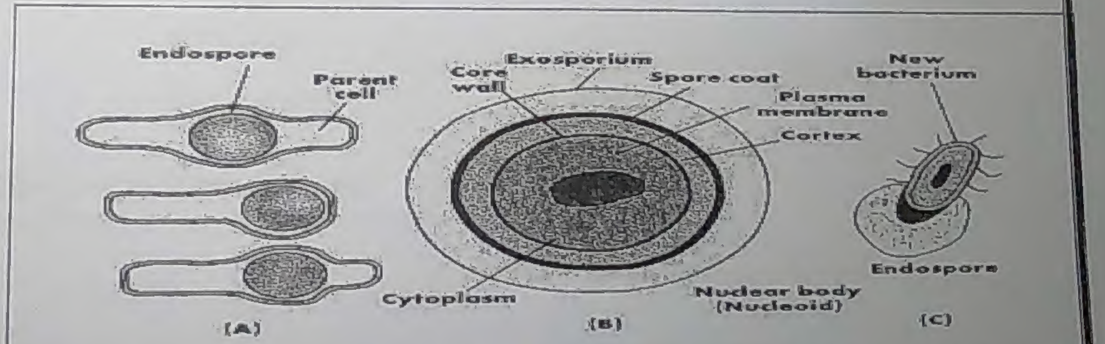
* اعملي رجيم - ناموجوه حيله و متخلفيش ....

- CM invaginates : enclosing section of cytoplasm
  - ♥ Chromosome
  - ♥ Some ribosomes
  - ♥ Other cytoplamic materials for germination
- Thick protective covering layer
  - ***i.Exosporium***
  - ***ii. Coat***
  - ***iii.Cortex***



3-Characters

- Absolute dormancy
  - ↳ metabolic inactivity } نوم
- No reproduction or growth



Endospore formation. A, Endospores according to their position in parent cells B, An edospore in cross-section. C, Germination of endospore

4 - Morphology

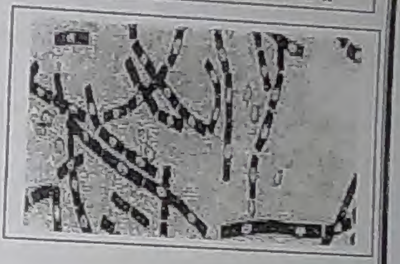
Position (in relation to bacillus)
- Central
- Subterminal
- Terminal

Shape
- Round
- Oval

Stain

Stained by ***special stain***

(Clear **uncolored** area in **stained** bacillus by **Gram**)



C - Viability & resistance

1-Factors leading to resistance

- High content of **Ca++ dipicolinate**
- Impermeability of **coat & cortex**
- **Low H_2O** content
- **Very low metabolic** & enzymatic activity

2-Resistant to

- Heating
- Disinfectants مطهرات [Alcohol]
- Dryness

3-Killing

By sterilization

D - Germination

- *Return to **vegetative form***
  - Can multiply
- When conditions become favorable (usually in **vivo**)

Compare and contrast between prokaryotic & eukaryotic cells

	Prokaryotic cells	Eukaryotic cells
1- Examples	Bacteria	Protozoa , fungi and algae
2-Nuclear material	*a.Primitive nucleus: nucleoid*	a.True nucleus
	b.Single chromosome	b.Multiple chromosomes
	c.No nuclear membrane	c.Nuclear membrane
3-Histones, nucleolus, mitotic apparatus	*Absent*	Present
4-Ribosomes	*30S , 50S & 70S* (polyribosome)	40S, 60S & 80S (polyribosome)
5-Mitochondria & microtubules	*Absent*	Present
6-Replication	??*Simple binary fission* انقسام ثنائي بسيط	Mitosis
7-Cytoplasmic membrane		
i.Cholesterol	*Absent* except in Mycoplasma	Present
ii.Mesosomes	*Present*	Absent
8- Cell wall		
i.Presence	*Present* except in Mycoplasma	Only in fungi
ii.Cause of rigidity	*Peptidoglycan*	Chitin

Essay questions on bacterial cell structure

1- Give a short account on peptidoglycan.

2- Mention functions of outer membrane.

3- Compare & contrast between ribosomes & mesosomes.

4- Compare & contrast between flagella &pili.

5- Compare & contrast between protein structures of bacteria (flagella & pili) → Pure Protein .

6 - Compare & contrast between capsule and spore regarding : site, structure &stain.

7- Mention 5 differences between prokaryotes & eukaryotes Vivo Vitro

8 - Give reason

a -Spores are highly resistant.

b-Rigidity of bacterial cell wall.

c-Bacterial cell membrane plays a role in disease production , respiration & chemotaxis.

→ distructive ①
→ Exotoxine ②

General Bacteriology 2

Bacterial Growth

Bacterial Growth

Definition

↑ in size & no of individual org.

Growth requirements

A-Nutritional requirements

1-Carbon & Nitrogen

According to C requirement , bacteria may be

Autotrophic

Require **inorganic** source of **C as CO_2**

to synthesize their organic metabolites

Heterotrophic (parasitic) – most pathogenic

Require **organic** source of **C from living host**

to synthesize their organic metabolites

According to N requirements

Exacting كثيرة المطالب → most pathogenic

Require **organic** source e.g a.a

Nonexacting

Require **inorganic** source e.g nitrates

2-Growth factors

a.a., purines & pyrimidines

B complex vitamins & blood

3-Inorganic ions (small amounts)

Phosphorus & sulfur

Ca++ & Mg++

B - Gaseous requirements

1-O_2 requirements

	1-Obligate aerobes	2-Obligate anaerobes	3-Facultative anaerobes	4-Microaerophiles
a-Growth	Only in **presence** of O_2	Only in the **absence** of O_2	In the **presence (better)** or **absence** of O_2	Only in the presence of **small amount** of O_2
b-Enzymes *i.Superoxide dismutase* *ii.Catalase*	**Present** ⬇ **Can degrade** toxic O_2 Metabolites : H_2O_2, O_2^- & OH^-	**Absent** ⬇ **Can't degrade** toxic O_2 Metabolites: H_2O_2, O_2^- & OH^-	**Present**	Present in **small amount**
c-Source of energy	**Aerobic** respiration	♥ **Anaerobic** respiration ♥ **Fermentation**	♣ **Aerobic** respiration ♣ Anaerobic resp.&**fermentation**	Aerobic respiration
d-Examples	Mycobacterium TB	Clostridia	**Most pathogenic bacteria**	Campylobacter

5-Aerotolerant anaerobes

Grow in the presence of O_2 (has **superoxide dismutase**) , but **don't use it** to obtain energy ⟶ **obligate fermenters**

TABLE 6.1 **The Effect of Oxygen on the Growth of Various Types of Bacteria**

	a. Obligate Aerobes	b. Facultative Anaerobes	c. Obligate Anaerobes	d. Aerotolerant Anaerobes	e. Microaerophiles
Effect of Oxygen on Growth	Only aerobic growth; oxygen required.	Both aerobic and anaerobic growth; greater growth in presence of oxygen.	Only anaerobic growth; ceases in presence of oxygen.	Only anaerobic growth; but continues in presence of oxygen.	Only aerobic growth; oxygen required in low concentration.
Bacterial Growth in Tube of Solid Growth Medium					
Explanation of Growth Patterns	Growth occurs only where high concentrations of oxygen have diffused into the medium.	Growth is best where most oxygen is present, but occurs throughout tube.	Growth occurs only where there is no oxygen.	Growth occurs evenly; oxygen has no effect.	Growth occurs only where a low concentration of oxygen has diffused into medium.

2-CO_2 requirements

Most bacteria need **only CO_2 present in air (0.05%)** ↳ most pathogenic ⟷ Some need **higher conc. of CO_2** (5-10%) e.g Neisseria

C-Physical requirements

1-Temperature

- ***Psychrophilic*** — Range : 5-30 C
- ***Mesophilic*** — Range : 10-45C
  e.g **Pathogenic bacteria** (OT:37C)
- ***Thermophilic*** — Range : 25-80 C

2- Hydrogen ion concentration (pH)

- ***Most pathogenic bacteria*** — 7.2-7.6 (**neutrophilic**)
- ***Lactobacillus*** — Acidic pH (**acidophilic**)
- ***Vibrio cholera*** — Alkaline pH (**alkalophilic**)

Measurement of bacterial growth

Bacterial count : measure n= of bacteria

Practical

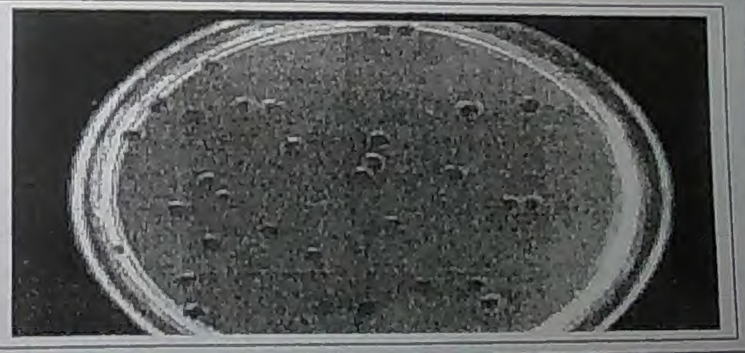
Total cell count : no of living & dead bacteria

- ***Dry weight***
- ***Turbidity***

Viable cell count : no of living bacteria

No of colony forming units (CFU)

Each bacterium multiplies ⟶ **1 colony**



✓ ***Generation time (doubling time)***

Time required by bacteria to **double its number**

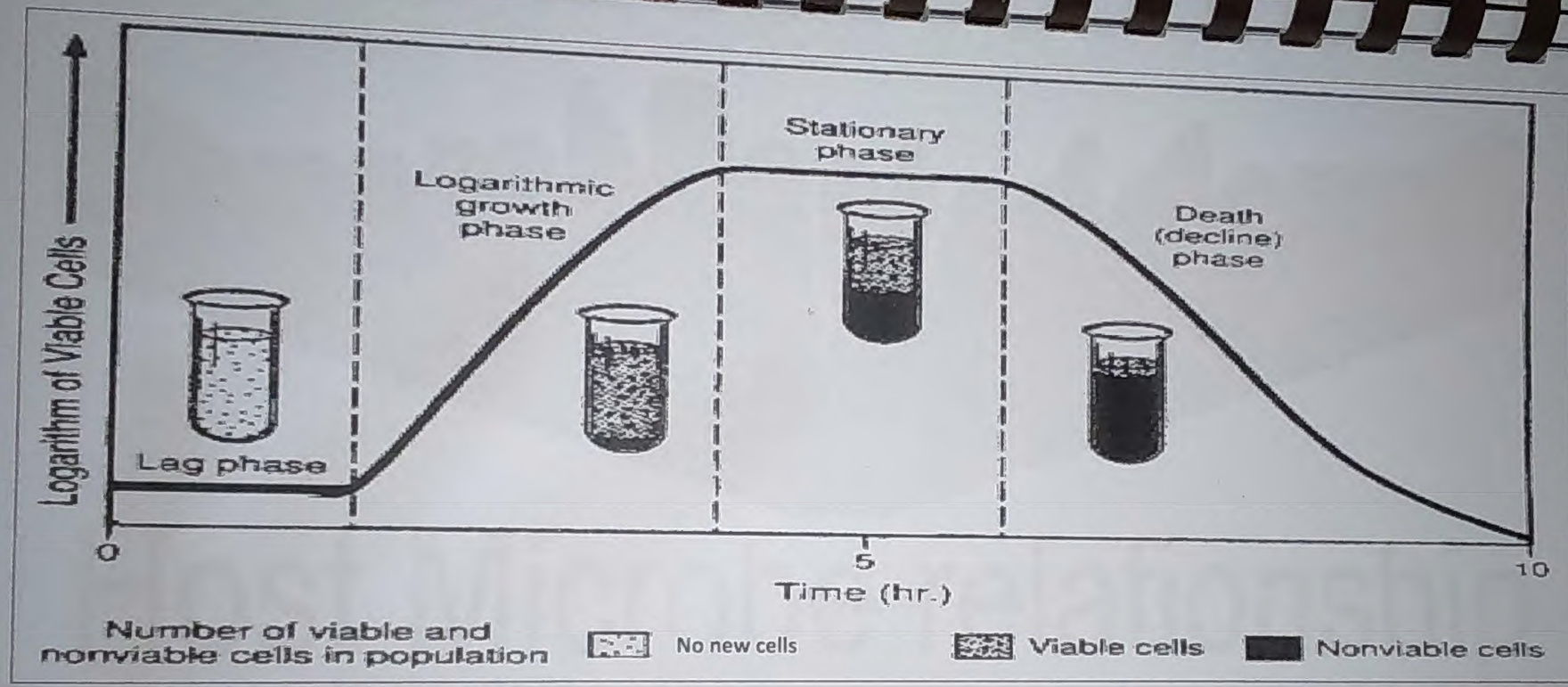
(varies from one species to another)

NB Fastidious bacteria (mycroplasma)

Bacteria requiring complex nutritional requirements

Bacterial Growth Curve

Lag phase (A)	Exponential (Logarithmic) Phase (B)	Stationary phase (C) (spore formation)	Decline phase (D)
1 - Etiology			
a.Bacteria adapt to new medium by forming enz. for growth. ***a. Its length depends on :*** i- Type of org. . ii.Type of growth medium iii.Size of the inoculum = no. of culture bacteria	*a.Most active phase of bact.* ↓ Highly sensitive to antibiotics ***b.This continues until:*** i- ↓ nutrients. ii- Accumulation of toxic metabolites.	↓ nutrients , O_2 starvation & *Accumulation of toxic metabolites* ↓ ↓ multiplication rate	Depletion of nutrients & *Maximal accumulation of toxic products* ↓ ↑ death rate
2 - No. of living bacteria (Viable count)			
No ↑ (but ↑ in size)	**Steady rapid ↑**	**Remains constant** (*n of dead = n of newly formed)*	**Rapid ↓** (culture may become sterile)
3 - Clinical significance (in vivo)			
Incubation period of ds	**Active disease** (signs & symptoms)		**Convalescence** & recovery



Essay questions

1-Compare & contrast between obligate aerobes & anaerobes.

2-Give reasons :

- Obligate anaerobes can't grow in presence of O_2
- Length of lag phase is variable
- Decline phase of bacterial growth

General Bacteriology 3

Host Microbe relationship

Bacterial Classification

Host Microbe relationship

Normal flora

A - DTT

Definition

Org.that grow in healthy persons

↓

Don't normally cause ds

Time of acquisition

Fetus is *sterile until rupture of membrane*

↓

Acquires flora (colonization) during passage in vagina

↓

Newborn acquires more flora from environment : food & other humans

Types

Resident

Found for prolonged time e.g in GIT

Transient

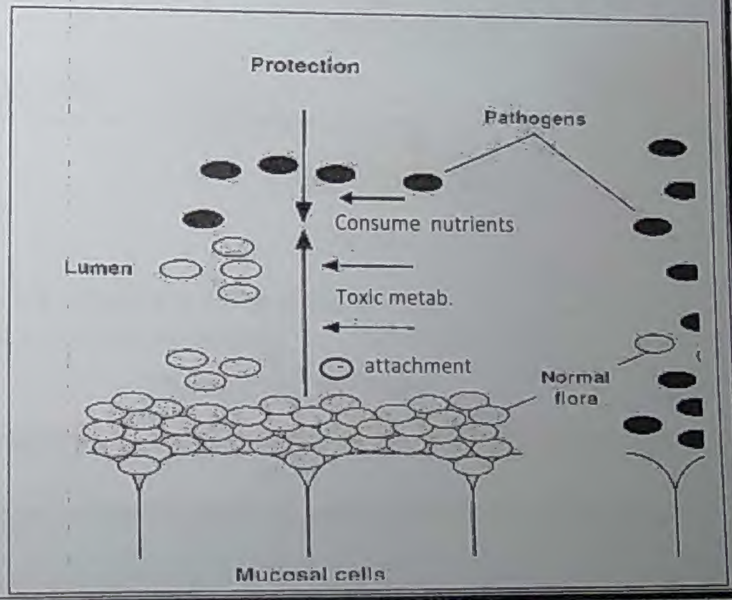
Found temporarily e.g in skin & nose

B - Beneficial effects : ⊙ pathogenic bacteria by

- Covering their attachement sites
- Consuming nutrients
- Production of toxic metabolites

C - Harmful effects : Oppurtunistic → cause ds

- In immune-compromised hosts
- When introduced outside their normal sites
  - ♦ E.coli of intestine
  - ♦ Staph.epidermidis of skin
- Superinfection (E)



Types of relationship between host & microbe

	Microbe	Host	Examples
1-Mutual	Benefits: Get food & energy from host	Benefits : gets Vit K & B	Some flora in large intestine
2-Commensal		Unharmed	Some flora in skin
3-Parasitic		Harmed	Pathogenic bacteria

Principles of infectious diseases

A- Steps of infection

Colonization

Establishment & multiplication of org.

on body surface without invasion

Infection

Establishment &multiplication **with invasion (parasitic relationship)**

- ***No disease*** → Inapparent or subclinical
- ***Disease*** (impairement of body function) → Infectious ds

B- Types of pathogens

- ***Primary :*** cause ds in healthy individuals
  e.g Diphtheria & TB bacilli
- ***Oppurtunistic***
  Flora

C- Factors affecting host parasite relationship

Host factors

Immunity

Natural & Acquired

Microbial factors

Pathogenecity of org.depends on their **virulence factors**

Virulence is the degree of pathogenecity of an org.

Bacterial virulence factors

Structure or product that enables bacteria to cause ds

I-Adherence to host cell → **Resistance of physical removal** → **Colonization**

Fimbria (pili)

Adhere to receptors on

GIT & urinary epithelium ⇔ RBCs

Non fimbrial adhesins in CW

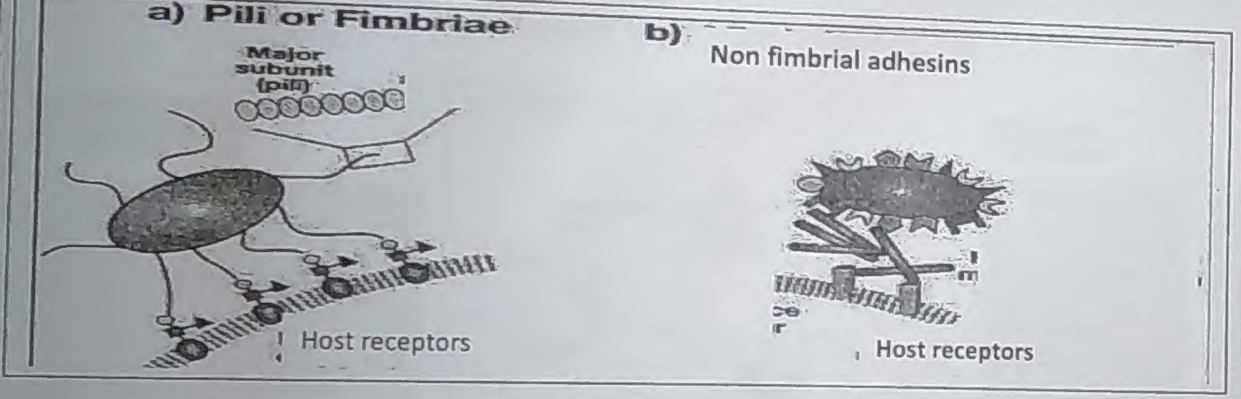
Bind to specific receptors

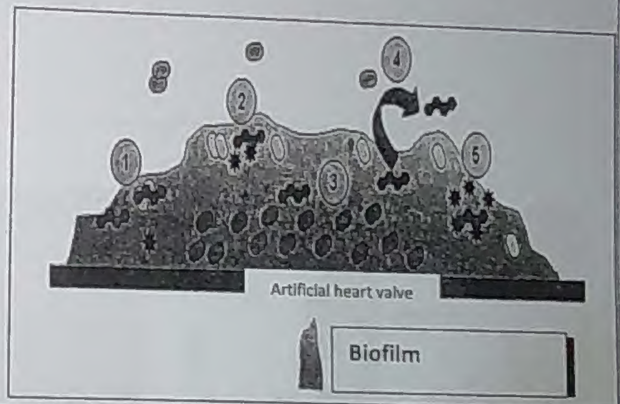
on host cell surface

Glycocalyx

↓

Biofilm formation





II-Invasion of host cells

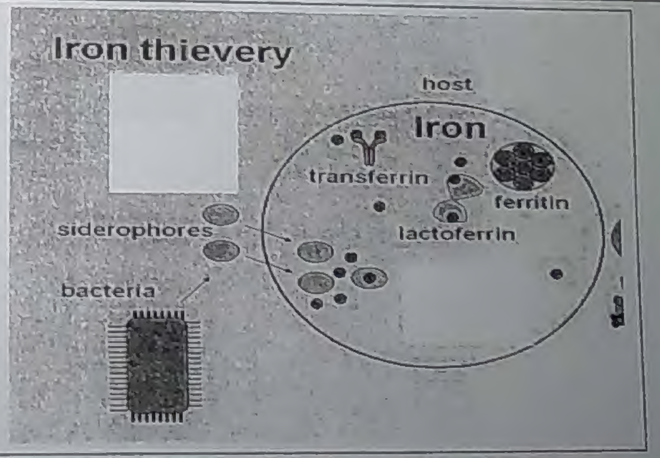
Entry in tissues → Multiplication → **Spread** to other tissues

III-Competition for human iron by Siderophores

Iron chelators excreted by bacteria into environment

↓

Bind iron → re-enter the bacterial cell



IV – Resistance to immunity

1-Antiphagocytic VF

Capsule

⊖ ***adherence to phagocytes***

- Strept.pneumoniae

Cell wall proteins

Bind to Fc of IgG

⊖ ***opsonization***

- Protein **A** of Staph.aureus
- Protein **G** of Strep.pyogenes

Coagulase

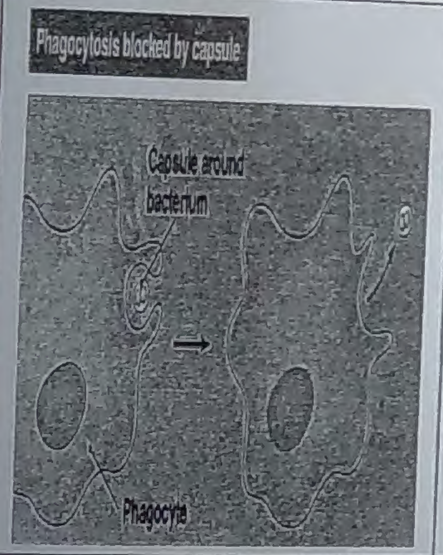
Forms ***fibrin clot*** from fibrinogen

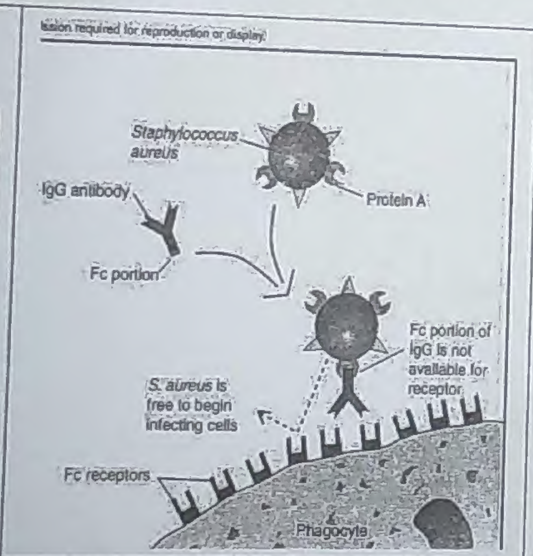
- Staph.aureus

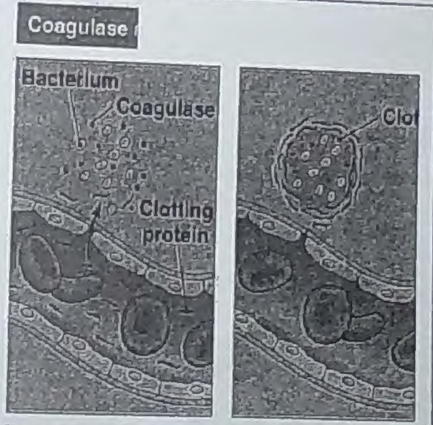
Leucocidin

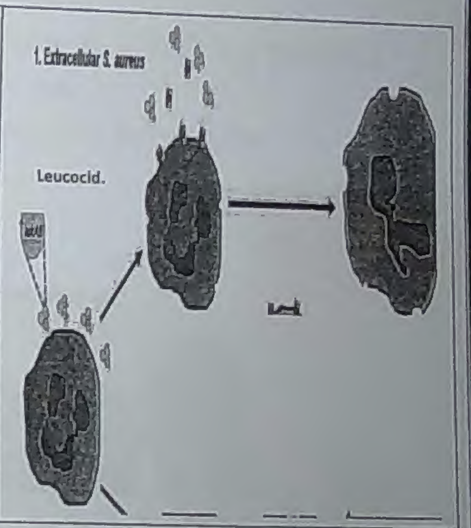
Kills phagocytes

- Staph.aureus









2-IgA protease

Degrades IgA ⟶ ***Adherence*** to mucosa

- Strept.pneumoniae

V- Bacterial toxins

A- Endotoxins (LPS)

Source

G –ve only

(part of CW)

Release

After

bacterial lysis

Effects

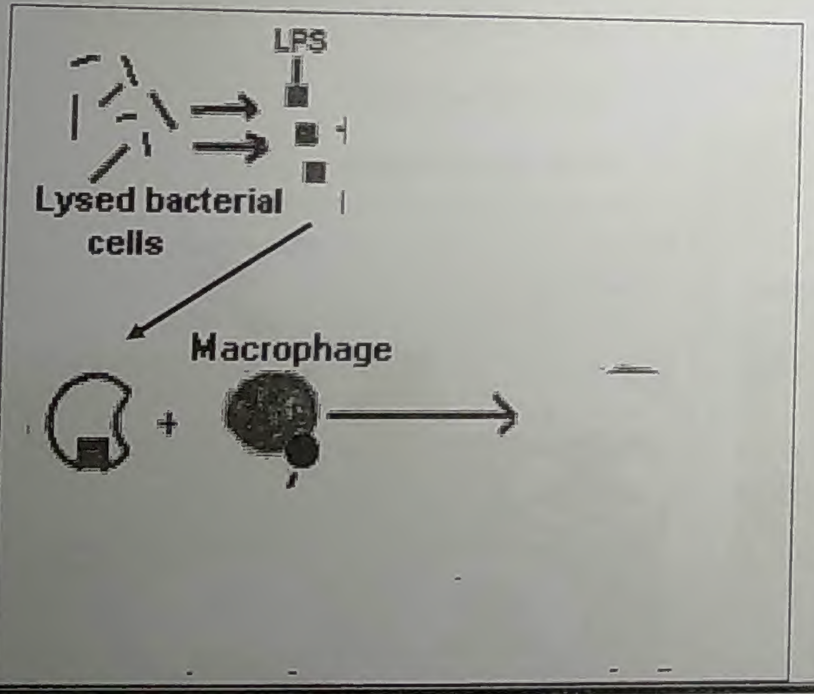
Systemic inflammatory response syndrome (SIRS)

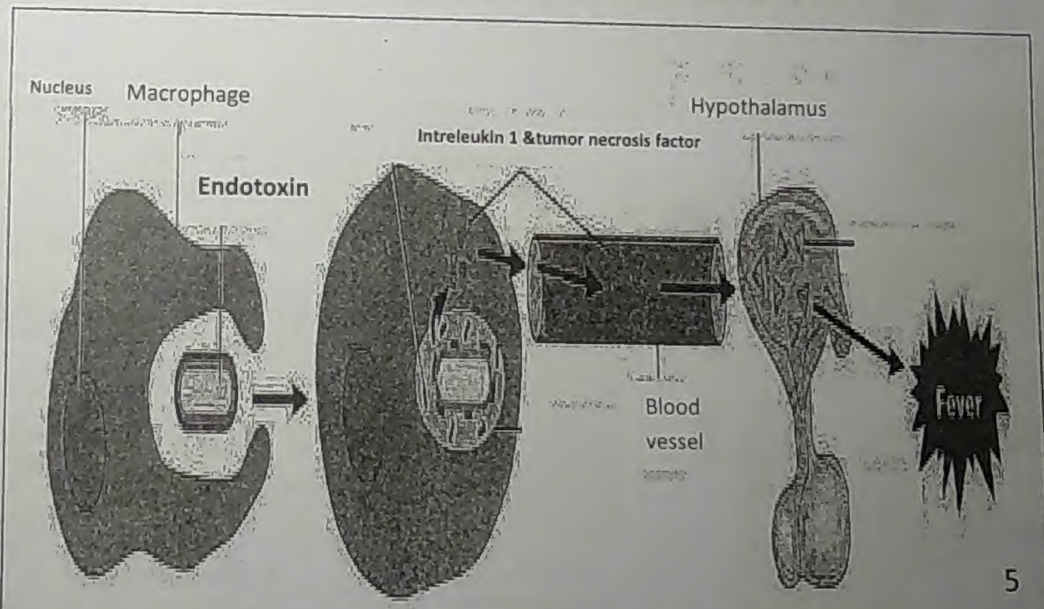
In severe systemic infection ,

high levels of LPS are released

↑ cytokines production from MQ e.g **TNF&IL1**

FEVER , SHOCK & DEATH





B – Exotoxins

1-Source
- G +ve
- G -ve

2-Release
- **Excreted during life** (6)
- After lysis

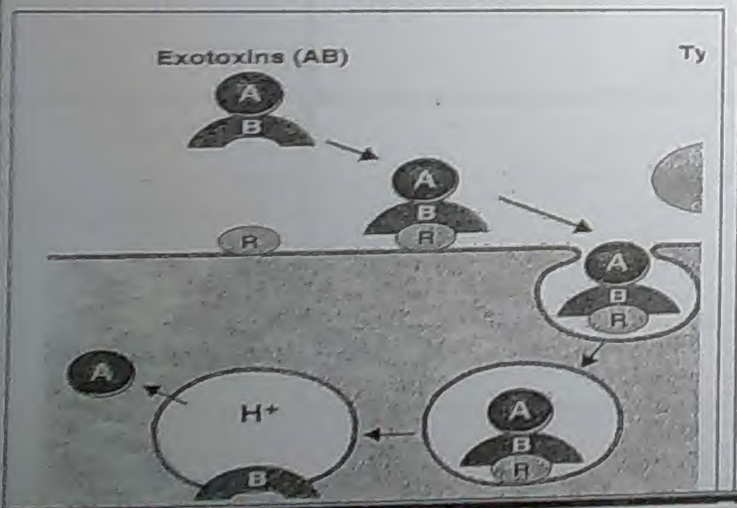
3-Target
- **Cyto** → Variety of cells
- **Specific**
  - Neuro
  - Hepato
  - Leuko

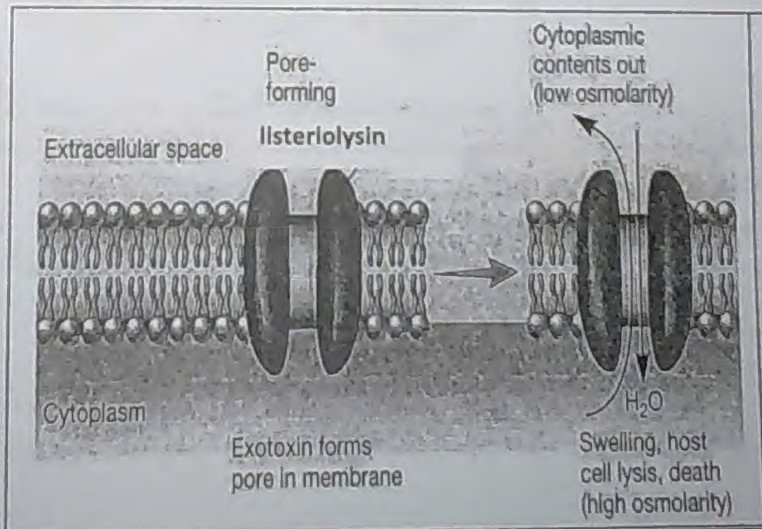
4-Structure &Functions (effects)

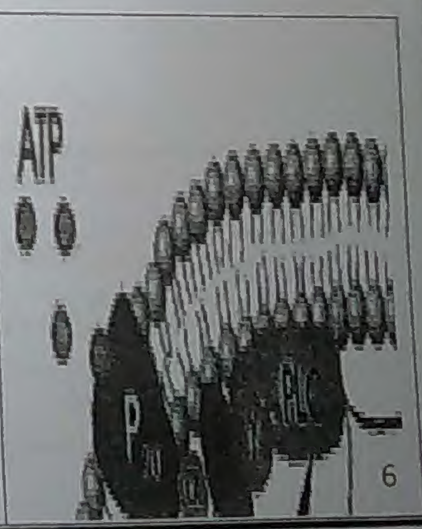
a. A-B toxins
- **Portion B** → ***Bind*** to specific cell receptors
- **Portion A** → ***Enzymatic (active)*** → Interferes with cell metabolism
  e.g diphtheria toxin

b. Membrane disrupting toxins (cytolytic enzymes)
- **Action**
  - ***Cyto*** → ***CM***
  - ***Disrupt***
  - ***Hemolysin*** → RBCs lysis
- **Types**
  - ***Pore forming*** → e.g listeriolysin
  - ***Lecithinase*** → (phospholipase C)

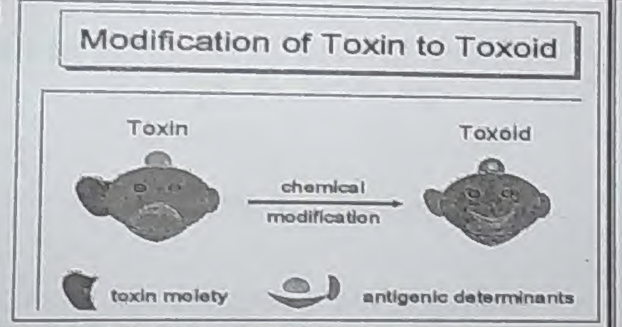
c. Super Ag







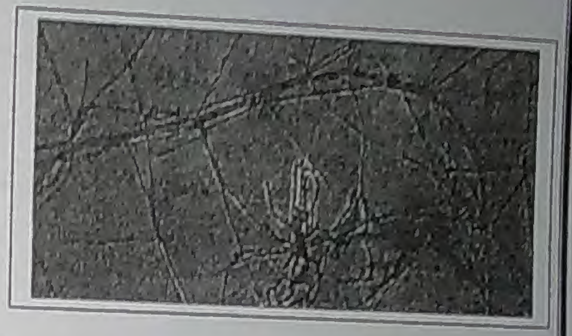
Comparison between exotoxin & endotoxin

	Exotoxin	Endotoxin
1-Source	Secreted by **both** G+ve (mainly) & G-ve bacteria	***Part of CW of G –ve only***
2-Release	**Diffusible :** secreted by living cell	Non diffusible *:* released on cell lysis
3-Structure	***Protein***	***Lipopolysaccharide***
4-Antigenecity	**High**	Low
5-Heat stability	Unstable to temp. > 60C	**Stable > 60C** for several hours
6-Effect of formalin	Convert it to **toxoid** ↓ Antigenic but non toxigenic 	No effect
7-Fever	No	**Yes,** by release of IL1&TNFα from MQ
8-Specificity	**Specific**	Non specific ↓ All cause fever & shock
9-Toxicity	Very high	**Low**

Bacterial Classification

I- Old system:

Based on *phenotypic characters*



A-Higher bacteria :

Actinomyces → Filamentous branching

B-Lower bacteria : simple unicellular org.

1-Reaction to Gram stain

i.G+ve : violet

ii.G-ve : pink

2-Shape

i.Cocci

ii.Bacilli

iii.Vibrio

iv.Sprilla

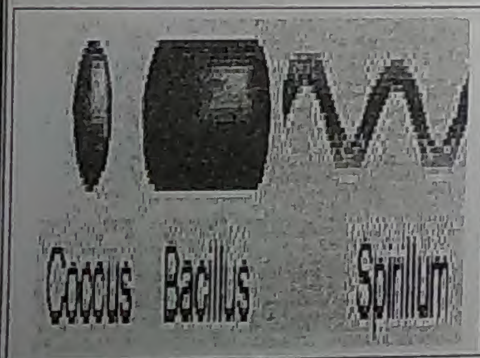
3-Nutritional requirements

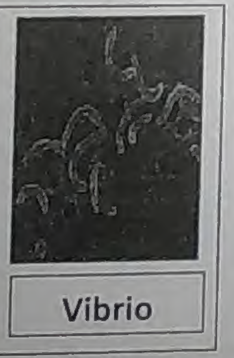
4-Methods of energy production

- **Aerobic respiration** — For aerobes
- **Glycolysis (fermentation)** — For anerobes

5-Pathogenecity

- **Saprophytes** — Live on **dead materials** soil, water, dust → *No disease*
- **Parasitic** — Live in body **of living creatures**
  - *1ry pathogens*
  - *0ppurtunistic flora*

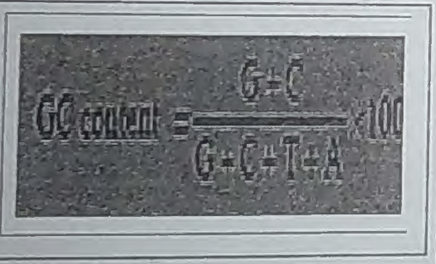
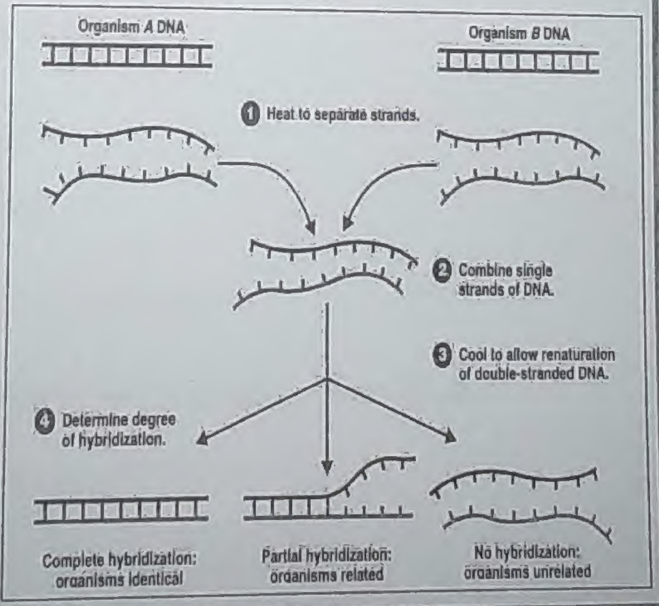
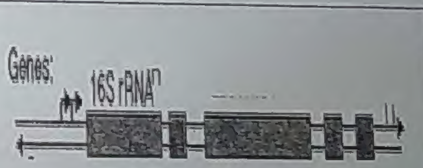




Vibrio

II-New system

Based on *genotypic characters* → determine *bacterial relatedness*

	Nucleotide base composition	**Nucleic base homology**	**Genome sequencing**
Principle	**Molecular % of G+C** in the total DNA	Homology of **DNA base sequences**	Nucleotide base **sequence analysis of rRNA genes**
Significance	***Fixed*** in strains of the ***same species***	Mixture of DNA from ***2 related species*** produce ***hybrid pairs***	Determine ***evolutionary relationships*** among bacteria
			

			Ancestor (from thousands of yrs)	
			Same sequence of gene coding	
Genus Staphylococci				**Genus X**
			Mixture of DNA produce	
Staph.aureus			*Staph.epidermidis*	
Strain 1	**Strain 2**	**Strain 3**		
Same				

Essay Questions

1- **Compare & contrast between exotoxins & endotoxins.**

2- **Give a short account on membrane disrupting toxins.**

3- **Give reasons :**

a. Normal flora are opportunistic pathogens.

b. Normal flora are considered as part of host immunity.

c. New system of bacterial classification determines bacterial relatedeness

d- How meningococci can cause septic shock ?

General Bacteriology 4

Bacterial Genetics

Bacterial Genetics

- BACTERIAL GENOME
  - Chromosome
  - Plasmid
  - Transposons
  - Bacteriophage
- BACTERIAL VARIATION
  - Phenotypic
  - Genotypic
    - •Mutation
    - •Gene transfer
- GENE
  - CLONING

Bacterial chromosome (Nucleoid)

A-Structure

- ♦Single
- ♦Circular
- ♦Supercoiled

- ♦ Double stranded DNA

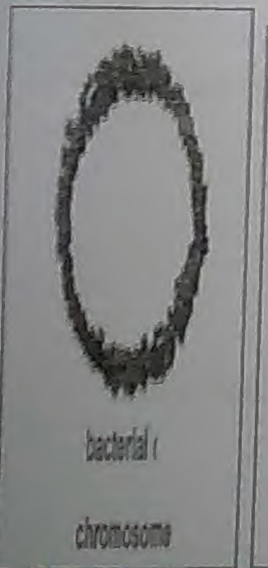
complementary to each other

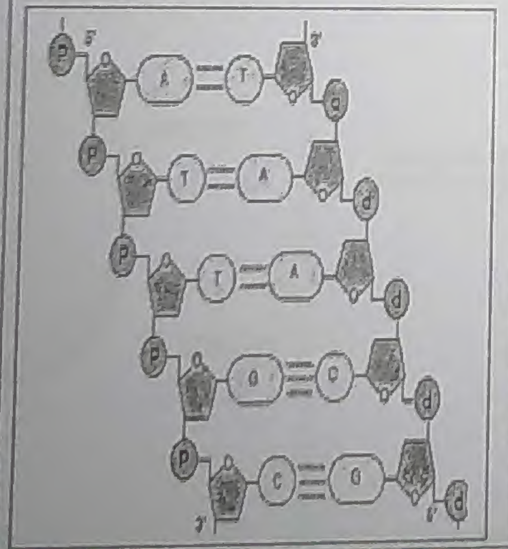
- •Guanine — Cytosine
- •Adenine — Thymine

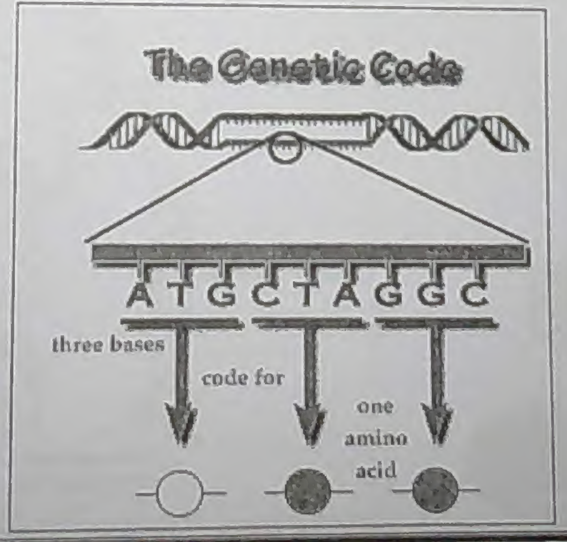
- ♦ Gene

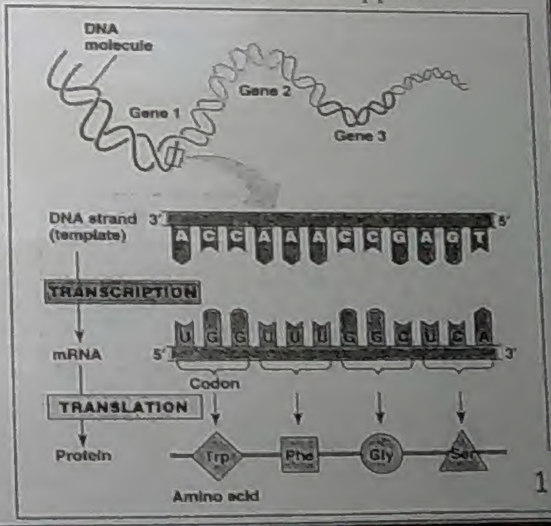
Segment of DNA carrying in its nucleotide sequence **information for a specific property**

- ♦NO
  - • Nuclear membrane
  - • Nucleolous, Histones
  - • Mitotic apparatus









B-Functions

Carries **essential genes** controlling properties & pathogenecity

C-Replication

Simple binary fission (semiconservative replication) :- each daughter cell recieve 50% of the ~~original~~ parents chromosomes

The 2 strands separate

▬

Each strand attaches itself to a **septal mesosome** & acts as a template

▬

Synthesis of complementary strand by **polymerase**

▬

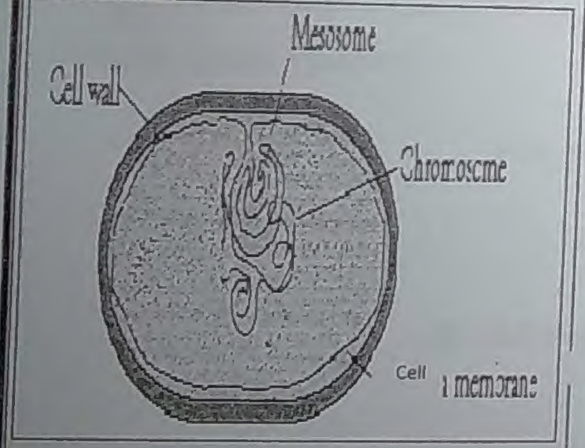
Inward growth of CM & CW

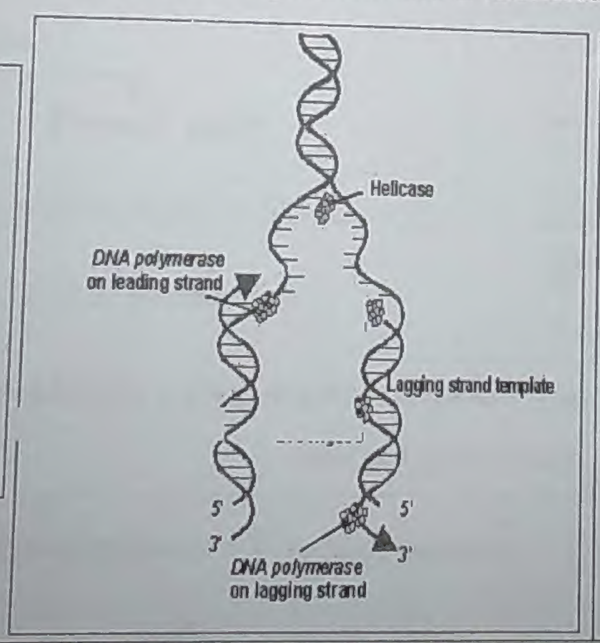
▬

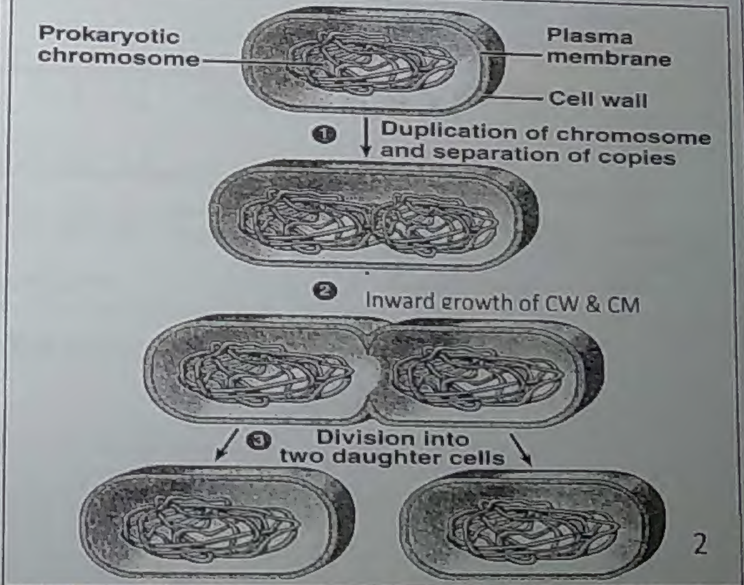
Protoplasm is divided into 2 equal parts

▬

Parent cell separates into **2 identical daughter cells** & identical to the parent.







Mutation

1- Definition

Change in nucleotide sequence along DNA molecule

2- Origin

Spontaneous

Replication error

Induced

Physical

- UV
- γ rays

Chemical

- ♦Alkylating substances
- ♦ Nitroso substances

3- Types

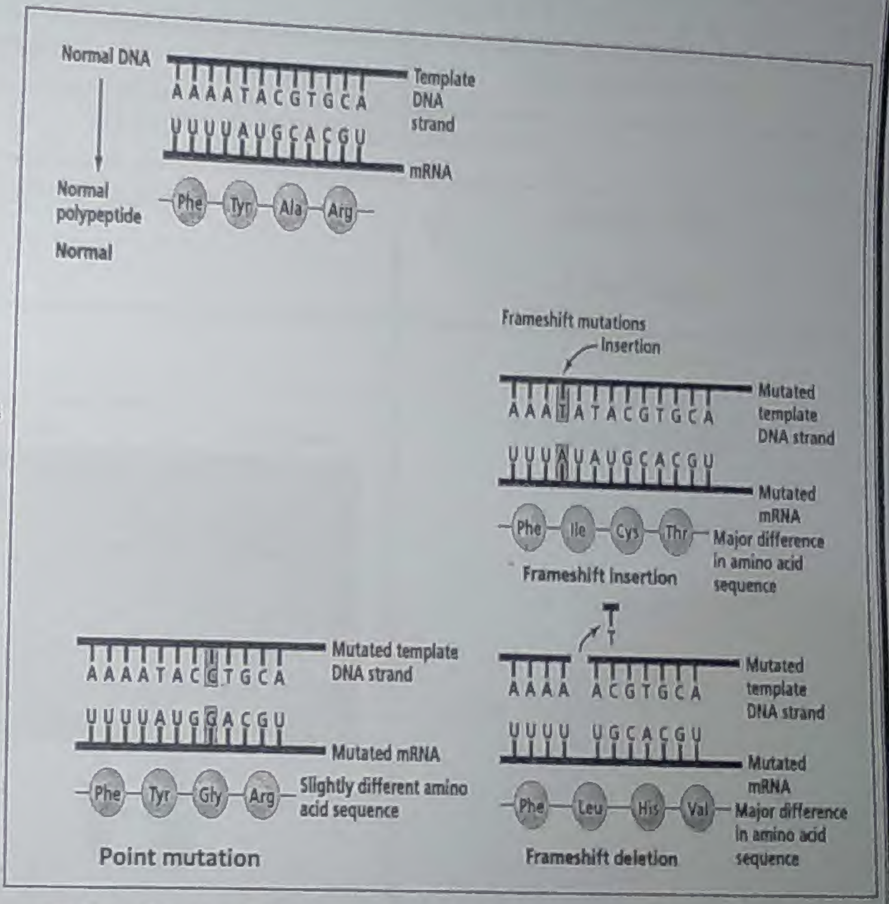
Single base (point)

Replacement of one nucleotide

Frame shift

Insertion or deletion of nucleotide

Shift in genetic code



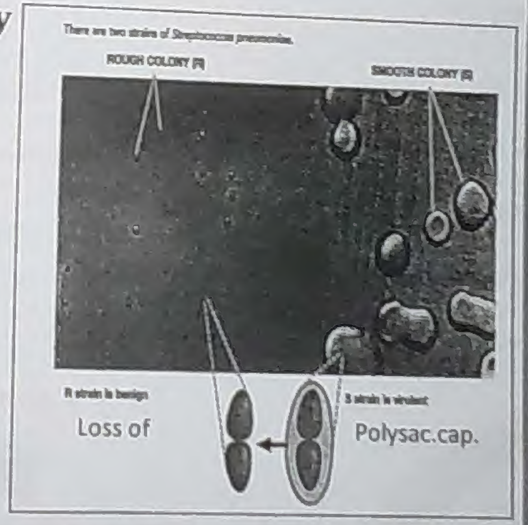
4-Medical importance of induced mutation

Getting mutant

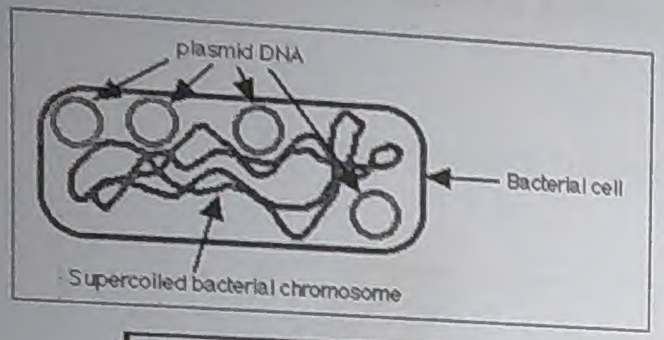
Of *low virulence* → used as vaccine

Producing *large amount of antibiotics*

Comparison between phenotypic & genotypic variation ★

	Phenotypic variation	Genotypic variation
1-Etiology	*Environmental changes*	*Changes in genes*
2-Effect	Changes in bacterial characters	
3-Characters	**i.Reversible** : when environmental cause is removed **ii.Non heritable**	**i.Irreversible** **ii.Heritable**
4-Examples	**i.Spore formation & vegetation** **ii.Change in colonial morphology** Bacterial growth on **unsuitable environment** ↓ ***Smooth to rough (S-R) variation***  **iii.↑ endopigment production** **by Staphylococci** If ***milk*** is added to the medium 	**i.Mutation** **ii.Gene transfer :** ♦ Conjugation ♦ Transformation ♦Transduction

Plasmids



Structure

Circular Ds DNA: ***Extrachromosomal*** & ***< the chromosome***

↳Double stranded.

Characters & Properties

Dispensable

Not essential for bacterial life

Autonomously ذاتي **replicate**

Replicate **independent of chromosome**

Many copies

of the same plasmid may coexist in same cell

Recombination اجتياح

Can **integrate** in chromosome

Episome ⇐MCQ

Transmissible

Transmitted to other bacteria by:

a.Conjugation

b.Transformation

c.Transduction

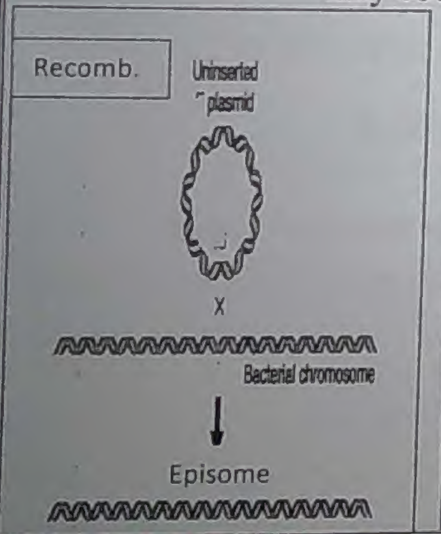
Self transfer (ST)

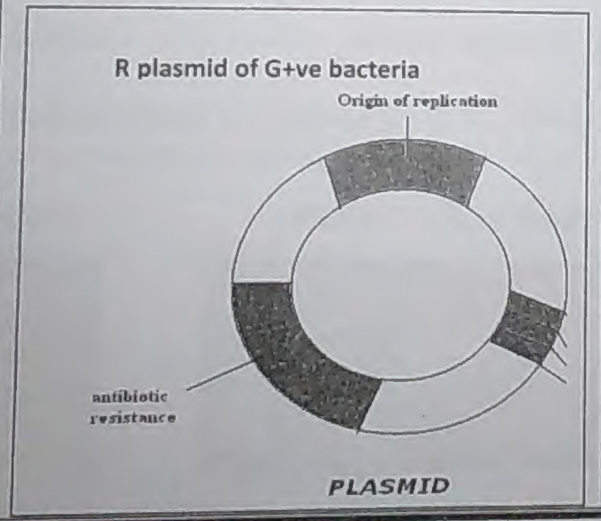
ST by conjugation

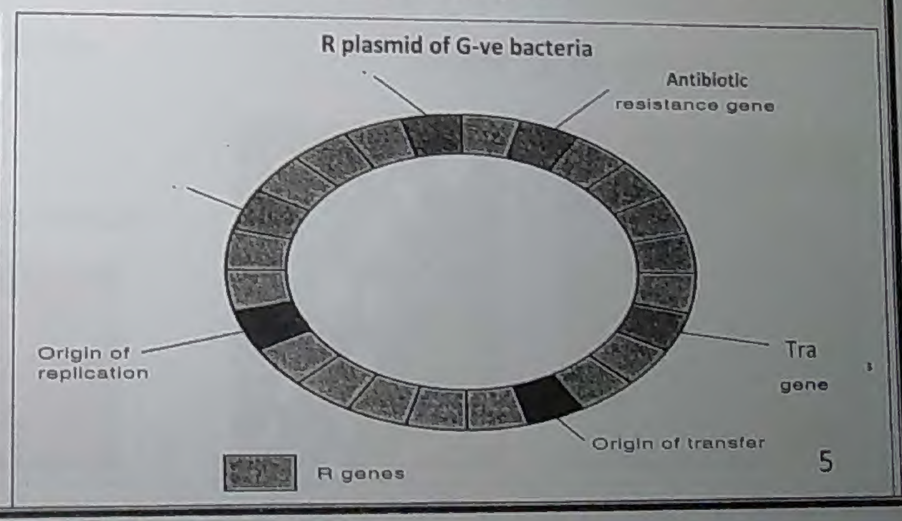
Plasmids of **G-ve** bacteria carry **tra genes** coding for **sex pilus**

No ST by conjug.

Plasmids of **G +ve** bact. has no tra genes







Cell properties determined by plasmids

1-Sex pilus formation

F plasmid carries tra genes → **sex pilus** → gene transfer by conjugation (only in G –ve)

↳ No Tra gene in any plasmid of G +ve.

2-Virulence plasmids TARI

- **Tox plasmids**: e.g coding for ***enterotoxin*** in E.coli
- **Adhesion plasmids**: e.g coding for ***pili*** in E.coli
- **Resistance to antibiotics: R plasmid**: e.g coding for ***β lactamase*** that degrades penicillins
- **Invasion plasmids**: e.g in Yersinia
  ↳ Coding for invasin protein for VF

3-Production of anti-bacterial substances

Bacteriocin (colicin) production : e.g Col plasmid of E.coli

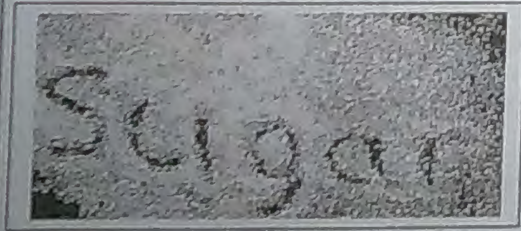
Antibiotic-like substance produced by certain bacteria to kill other bacteria of ***same or closely related species***

Antibiotic production
↳ It kills related & unrelated species.

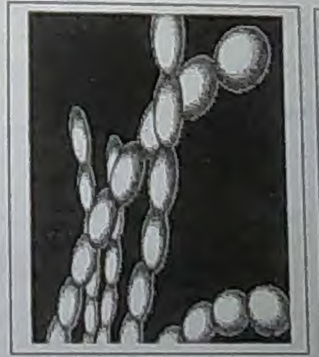
e.g Plasmid in ***Streptomyces*** coding for ***streptomycin***

4-Biochemical activities (In Vitro)

Sugar fermentation



Resistance to heavy metals



Conjugation : gene transfer by sex pilus (only in G - ve)

Transfer of F plasmid

(Fertility or sex plasmid)

Sex pilus of donor F+ cell

comes in contact with recipient F- cell

⬇

Endonuclease cleaves 1 strand

of F plasmid at origin of transfer

⬇

Unwinding of 2 strands of F plasmid

by helicase enzyme

⬇

1 strand passes through sex pilus to F- cell

⬇

Complementary strand is formed by both cells

By Polymerase

⬇

F- recipient cell is changed to F+ cell

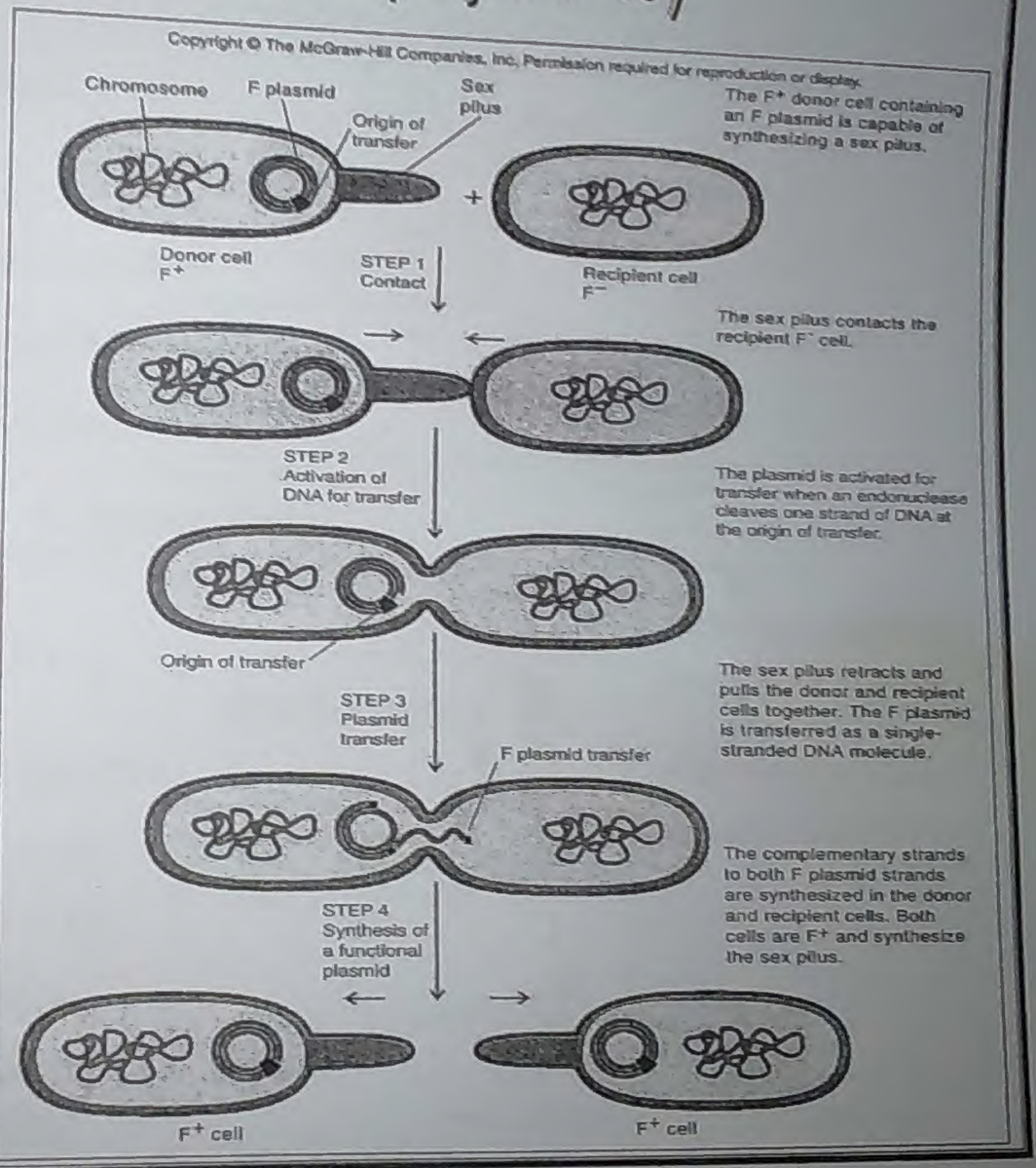
NB

Plasmid coding for penicillinase in Staph.(G+ve)

can't be transferred by conjugation

⬇

Transferred by transduction



Transformation

A-Definition

Uptake of ***free naked DNA*** (part of chromosome or plasmid)

B-Mechanism

Dead bacteria release DNA

↓

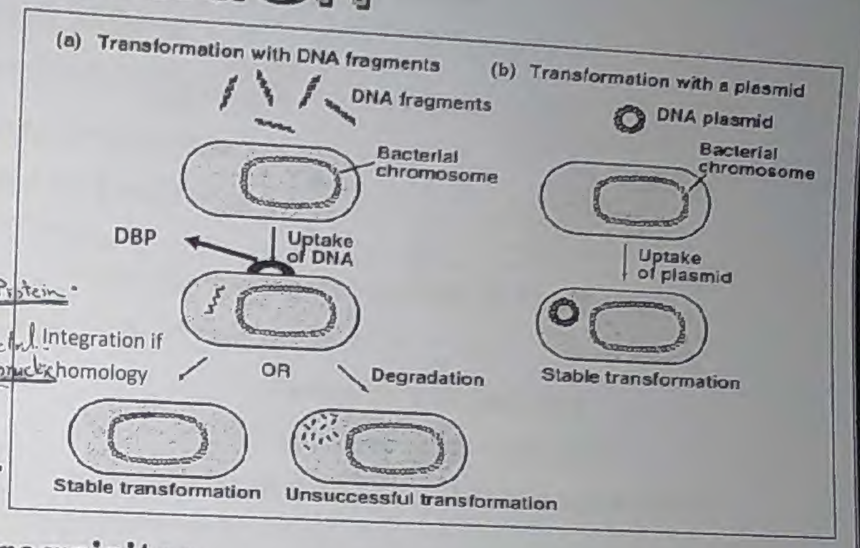
Binds to **competent** recipient bacterium

↓

Recombines with its DNA (or remains free if it is plasmid)

↓

New character if DNA carries new gene e.g **virulence factor.**



C-Prerequisites

Competence

Natural competence is uncommon

due to presence of

restriction endonuclease

which digests

foreign DNA

Artificial competence

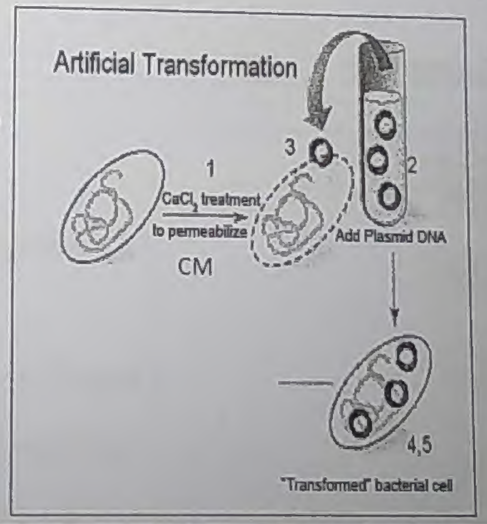
Treating cell with

$CaCl_2$ or heat shock

↓

↑ CM permeability

(Used in **genetic engineering**)



Homology between donor & recipient

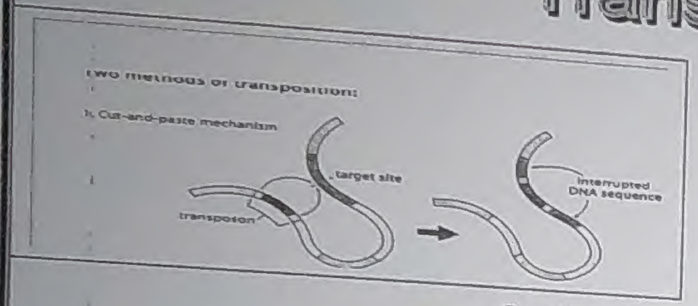
Absence of homology

prevents recombination

↓

DNA is degraded

Transposons : jumping genes

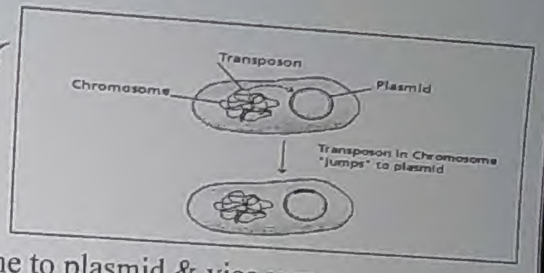


A-Structure & Effects

Genetic elements (several kbp) Kilo bar pair

that can **move** *(jump or transpose)*

from **one location to another**

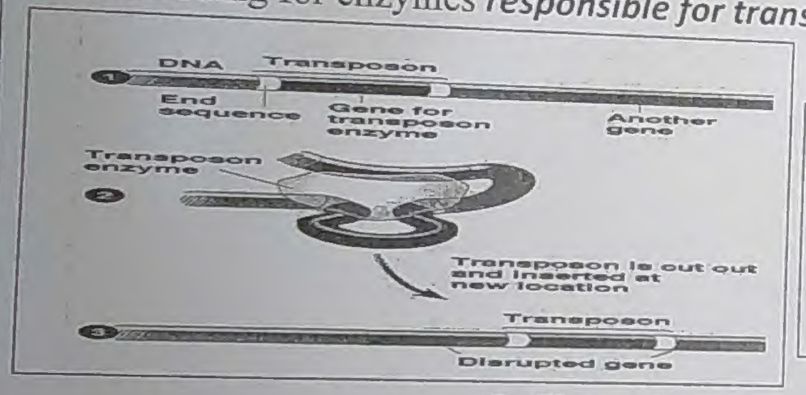


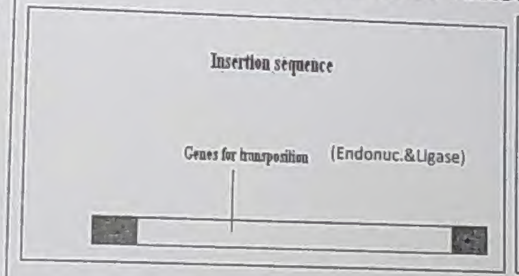
On the same chromosome ⟷ From chromosome to plasmid & vice versa

B-Types

Simple insertion sequence

Genes coding for enzymes *responsible for transposition*

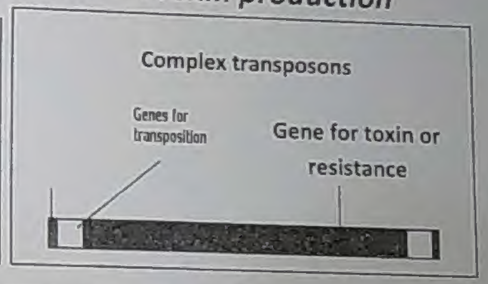




Complex transposons

As before + gene coding for

♦ ***Antibiotic resistance*** ♦***Toxin production***



C- Characters

Can't autonomously replicate

Must replicate **within a *replicon***

(self replicating DNA)

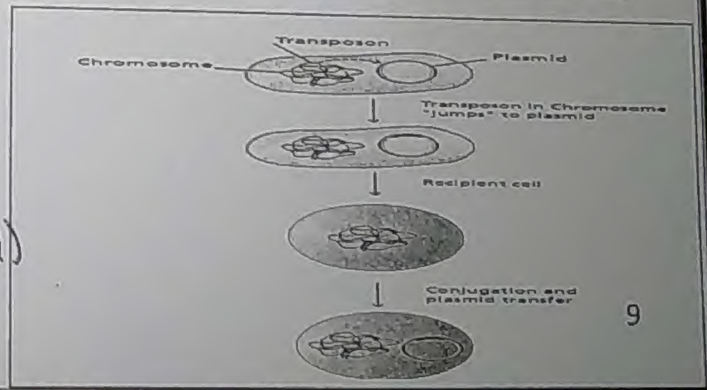
Chromosome ⟷ plasmid

Transmissible : by conjugation

If inserted into a

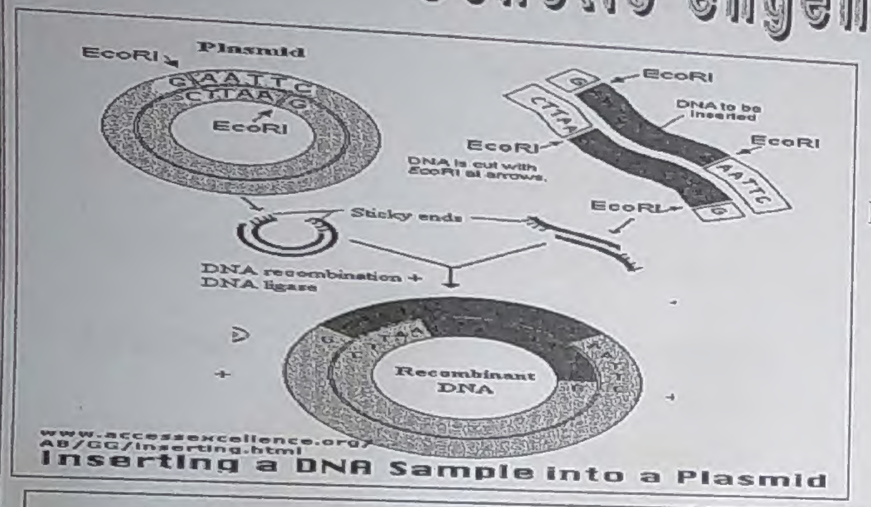
transmissible plasmid (F Plasmid)

Disseminate through bact.population

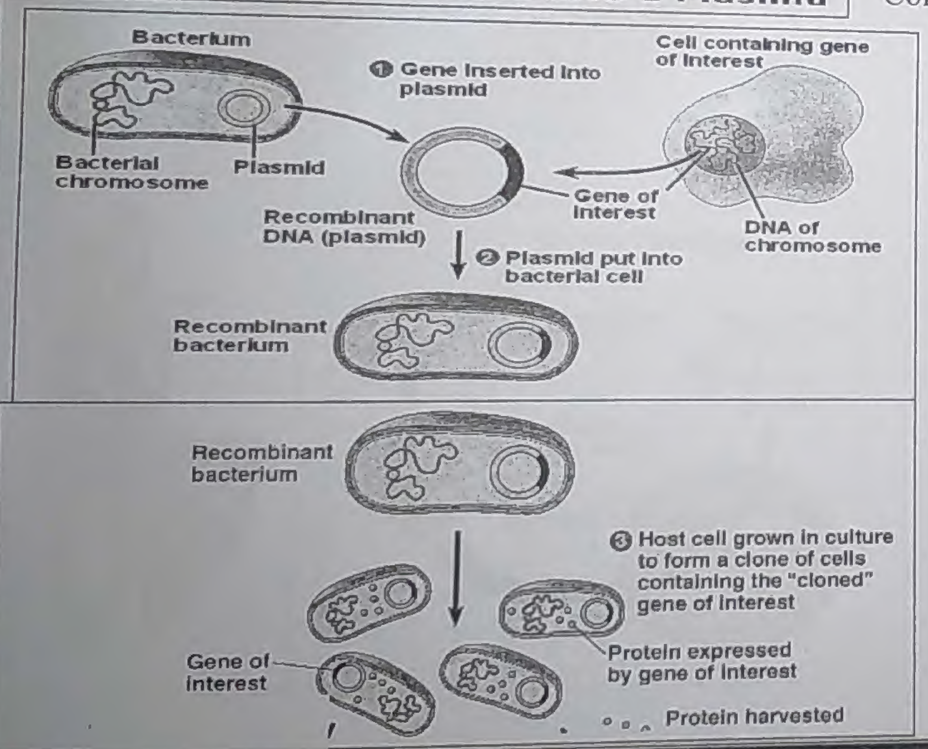


NB:- Transposons isn't ~~duplicate~~ in cytoplasm replicone

Genetic engeneering (gene cloning)



Inserting a DNA Sample into a Plasmid



A-Technique

1-Extraction of

DNA containing **the specific gene** — from human or animal

Vector (plasmid or phage) — from bacteria or yeast

2-Specific DNA & vector are

Treated with **restriction endonuclease**

Complementary (cohesive or sticky) ends

Recombinant DNA molecule

Transferred into bacterial cell or yeast (host cell)

by **transformation**

3-The cell multiplies

Creation of **many clones**

(genetically identical bacteria)

Each produces **the gene product (a protein)**

Harvesting the protein

from culture containing the clones

B-Applications

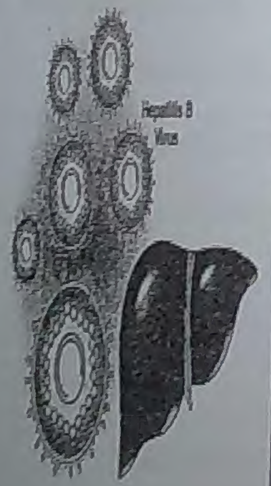
Production of

i.Hormones

e.g insulin

ii.Vaccines

e.g Hepatitis B



Gene therapy

Preparation of

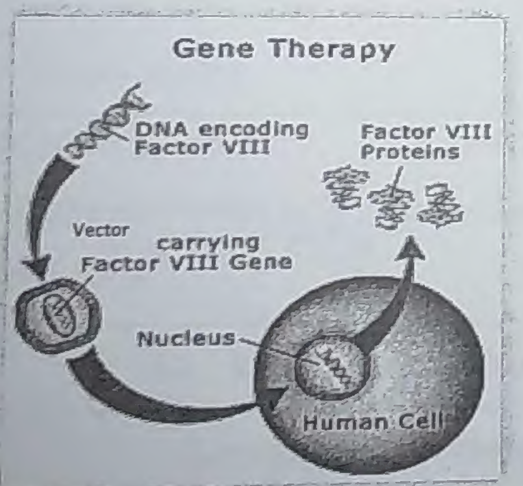
cloned normal gene

↓

Insertion into human cell

↓

Correct genetic disorder



Diagnosis of infectious diseases

Addition of ***NA probe***

(SS DNA or RNA labeled

with fluorescent dye,

radioactive isotope or enzyme)

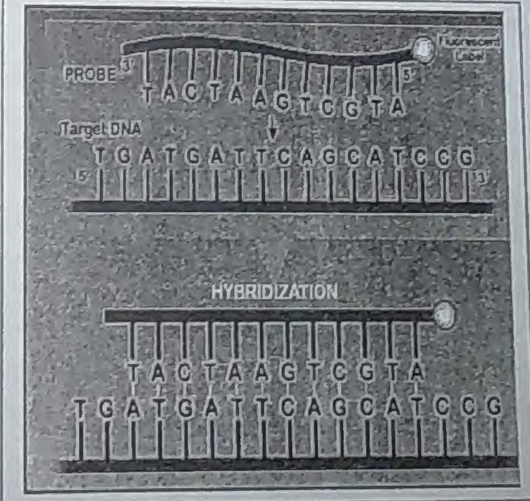
↓

Detects **complementary sequence**

of microbial gene

↓

Hybridization

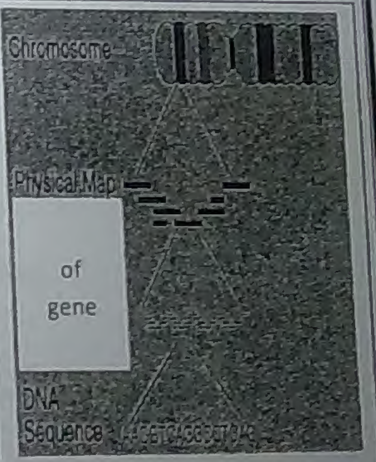


Chromosomal mapping & DNA sequencing

Determination of

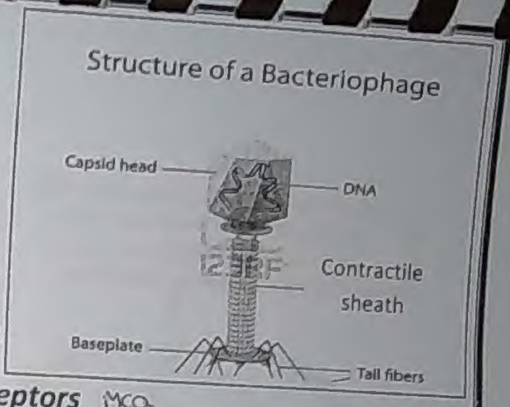
gene location

in org.'s genome



Bacteriophage

VIRUS THAT INFECTS BACTERIA



A-Structure

Head

Protein **capsid** containing **DNA** (mainly) or RNA

Tail

Hollow core surrounded by **contractile sheath**

Base plate ending in **tail fibers**

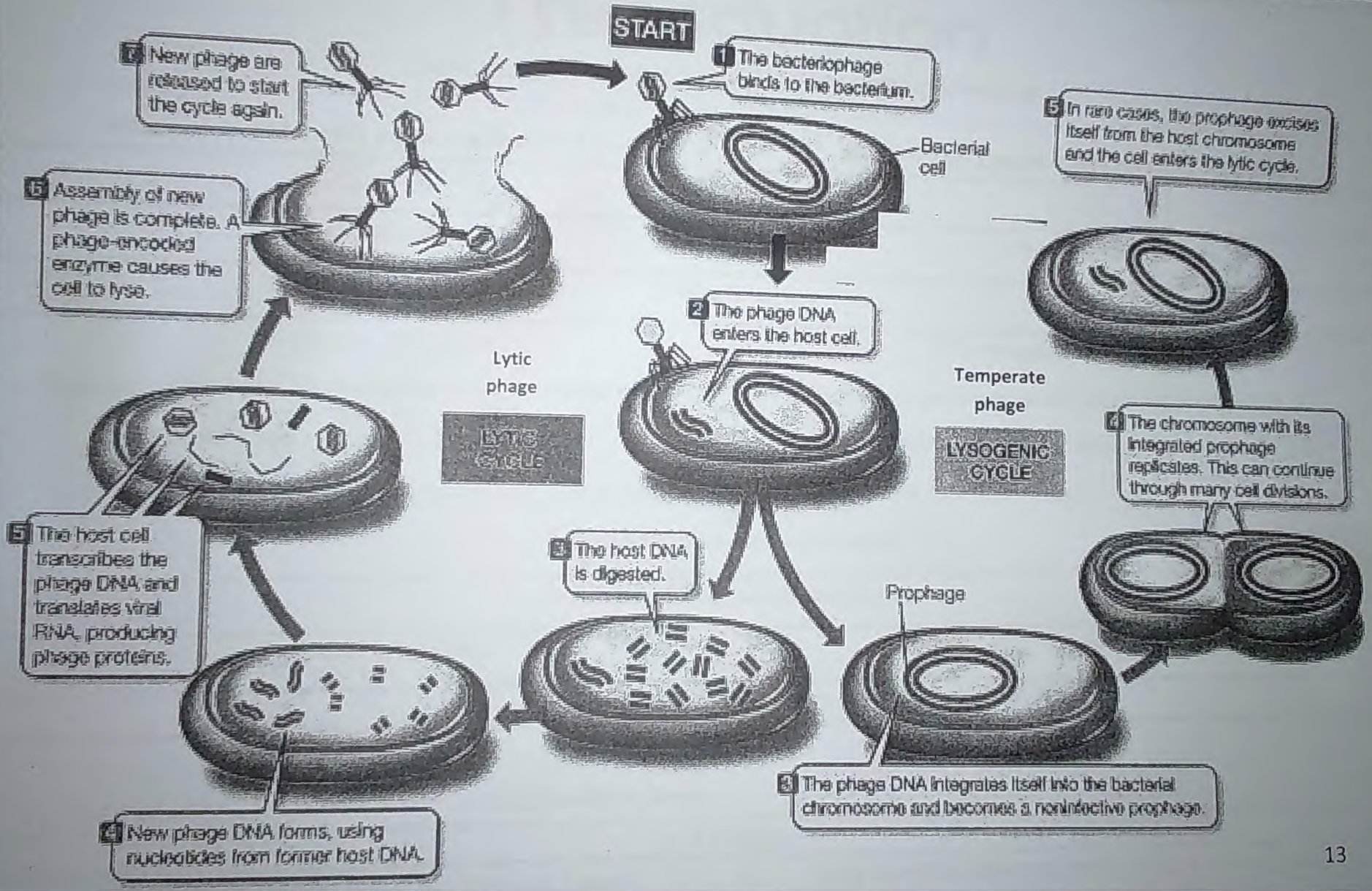
Attachment to specific CW receptors MCQ

B-Interaction between Bacteriophage & Bacterial host

Lysis	Lysogeny مميد بالقتل
Lytic (virulent) phage infects bacteria virulence gene ← are active ↓ Remains **extrachromosomal** ↓ Multiplies immediately → bacterial **lysis** ↓ Release of newly formed phages	virulence gene are inactive **Temperate** معتدل phage infects bacteria ↓ **Integrates** in bacterial chromosome, cd "***prophage"(non lytic)*** ↓ **No lysis** (no multiplication) ↓ Replicates with bact.chromosome & transferred to progeny
NB. Lysis may occur early due to **adsorption of large no of phages (heavy inf.)** ↳ rupture of cell membrane onto bacterial cells	Bacteria may acquire **new properties due to phage own genes** e.g **Toxin production** by C.diphtheria & Strept.pyogenes (eryhrogenic toxin) This is cd ***: "Lysogenic conversion"***

C - Importance of bacteriophages

Transduction	**Lysogenic conversion**	**Genetic engineering**	**Phage typing**
Gene transfer between bacteria	(E)	Act as vectors	Identification of bacteria

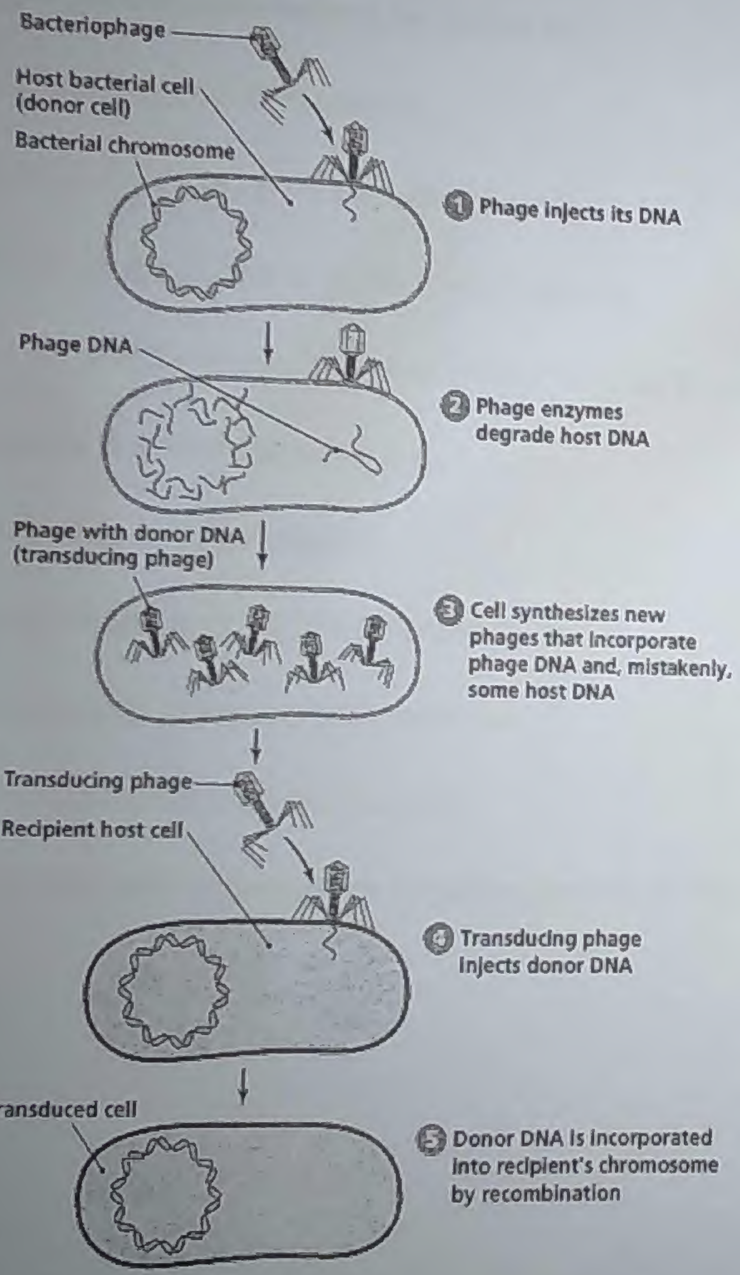

START
1 The bacteriophage binds to the bacterium.
Bacterial cell
2 The phage DNA enters the host cell.
Lytic phage
LYTIC CYCLE
3 The host DNA is digested.
4 New phage DNA forms, using nucleotides from former host DNA.
5 The host cell transcribes the phage DNA and translates viral RNA, producing phage proteins.
6 Assembly of new phage is complete. A phage-encoded enzyme causes the cell to lyse.
7 New phage are released to start the cycle again.
Temperate phage
LYSOGENIC CYCLE
Prophage
5 In rare cases, the prophage excises itself from the host chromosome and the cell enters the lytic cycle.
4 The chromosome with its integrated prophage replicates. This can continue through many cell divisions.
3 The phage DNA integrates itself into the bacterial chromosome and becomes a noninfective prophage.

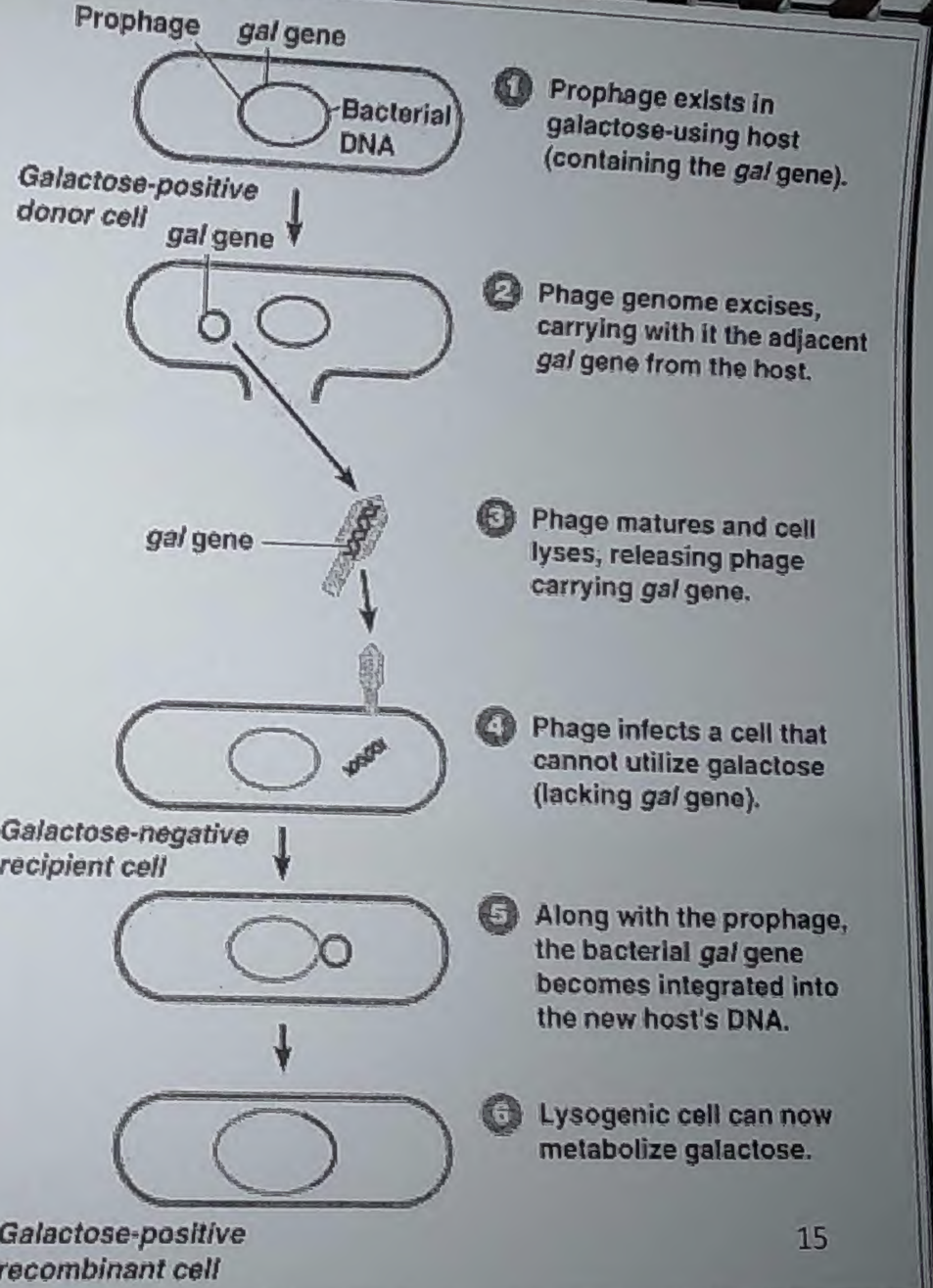
Transduction

Gene transfer between bacteria by **BACTERIOPHAGE**

Generalized transduction	**Specialized transduction**
1-Type of phage	
Lytic ~~or temperate~~	Temperate
2-Mechanism	
During the lytic cycle, a fragment of bacterial chromosome is **occasionally enclosed in the head** of a newly formed phage ↓ The transducing phage transfers this fragment into **a new bacterium** ↓ ***Recombination (crossing over)***	In a **lysogenic bacterium** , the prophage occasionally separates **incorrectly** & carries a **specific fragment** of adjacent chromosomal DNA ↓ The transducing phage transfers this fragment into **a new bacterium** ↓ ***Recombination (crossing over)***
3-Transducing phage contains:	
Only **bacterial** DNA	Bacterial DNA **+ part of phage** DNA
4-Transfer of plasmid	
Yes e.g Plasmid coding for ***penicillinase (β lactamase) in Staph***	No
5-Name	
Any part of bacterial DNA has an equal chance to be transduced	***Only specific adjacent part*** to phage has a chance to be transduced

اكتساب صفات جديده والبكتيريا مهددة بالقتل





Essay questions

1- Cell properties determined by plasmids.

2- Structure & types of plasmids.

3- Structure & importance of bacteriophage.

4- Medical applications of genetic engineering.

5- Mention 2 differences between bacterial lysis & lysogeny by phages.

6- **Compare & contrast between**:

   a. Plasmids & transposons.

   b. Specialized & generalized transduction.

   c. Phenotypic & genotypic variation.

   d. Conjugation & transformation.

7- Explain the role of prophage in pathogenesis of Diphteria.

General Bacteriology 5

ANTIMICROBIALS

Antimicrobials

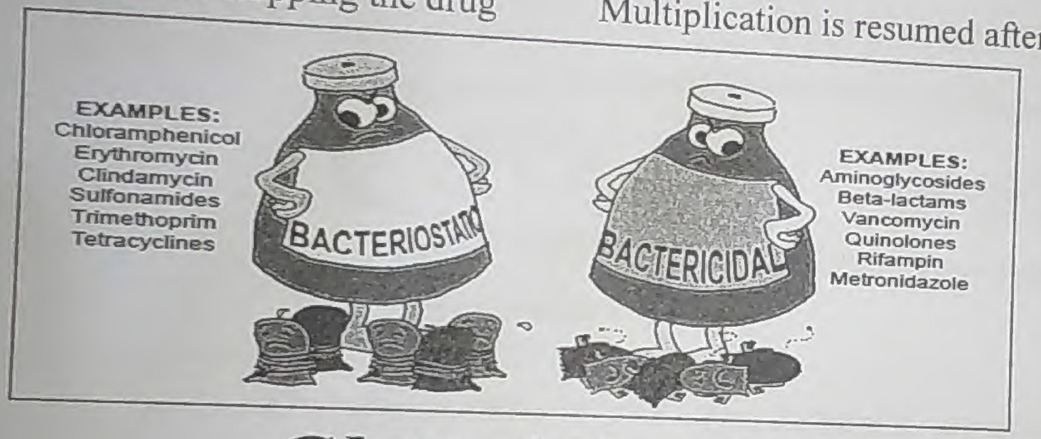
Substances that **kill** → **Cidal** → No multiplication after stopping the drug

OR

⊖ ***growth & multiplication*** of organisms → ***Static*** → Multiplication is resumed after stopping the drug

In vivo

Can be used systemically



Classification of antibiotics

According to origin

Naturally occuring

2ry metabolites of ***living org.***

Bacteria
- ♥ Streptomyces
- ♥ Bacillus

Molds
- ♣ Penicillium
- ♣ Cephalosporium

Chemotherapeutics

Synthesized in laboratory

According to spectrum

Broad

On ***both*** Gram +ve & Gram -ve bacteria

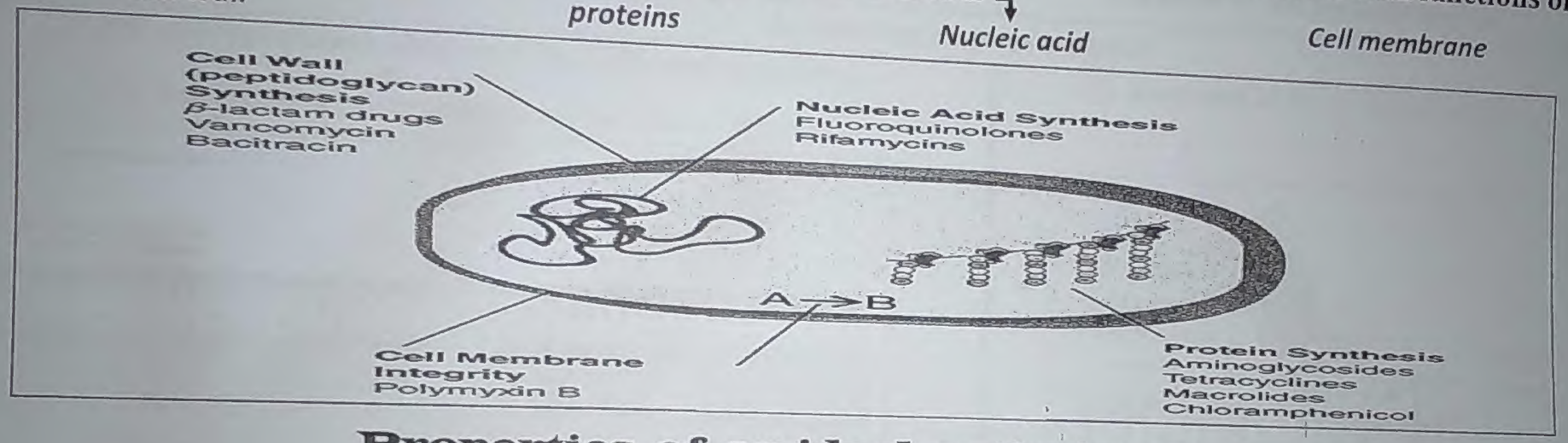
Narrow

On ***either*** of them only

Mechanisms of action of antimicrobials

⊖ Synthesis of

- Cell wall
- proteins
- Nucleic acid
- Interfere with functions of Cell membrane



Properties of an ideal antimicrobial

A – Bacteriological

- ***Selective toxicity***
  - ⊖ structure or metabolic channel
  - present in bacteria but **not in host cell** e.g CW
  - Toxic to org. not to host
- ***Cidal***
- ***Broad spectrum***
- ***Not subjected to bacterial resistance***

B – Pharmacological

- ***No*** allergy
- ***Oral*** administration & ***H_2O*** soluble
- ***Effective level*** in all BFs
- ***No affection*** by BFs
- ***Slow excretion***

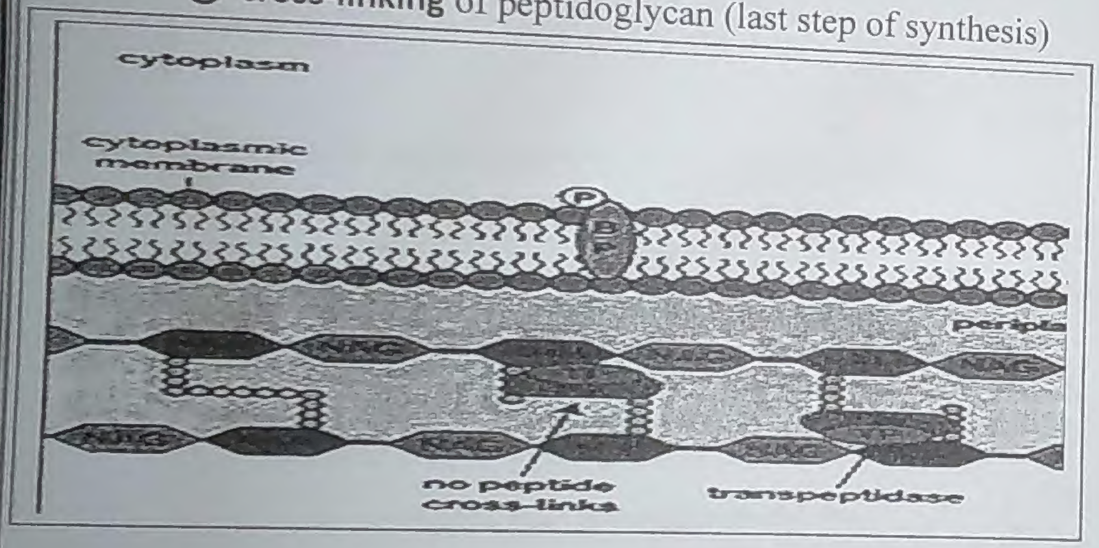
Antibiotics acting on CW

β lactam compounds (contain β lactam ring) | **Other CW inhibitors**

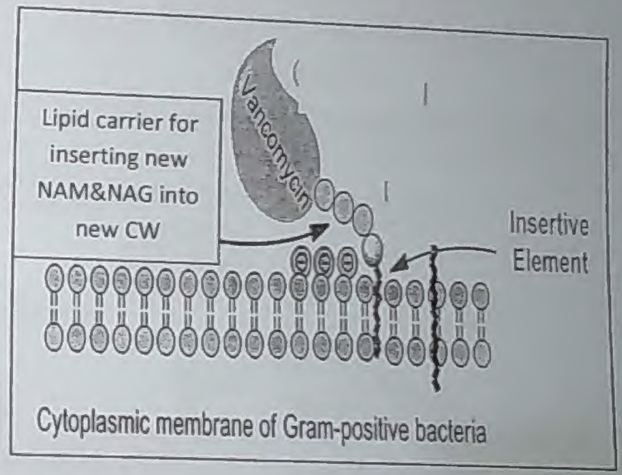
A-Mechanism of action

⊖ **transpeptidase enzymes** (Penicillin binding proteins)

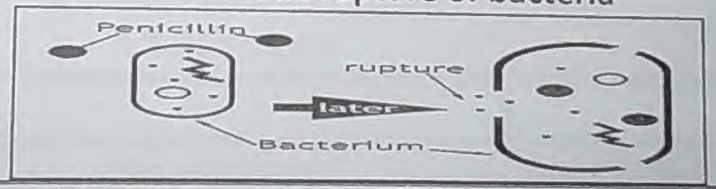
⊖ **cross-linking** of peptidoglycan (last step of synthesis)



⊖ **early step** of PG synthesis in CM



Osmotic rupture of bacteria



B- Advantages : **High selective toxicity**; no harm to human cells which have no CW

C-Disadvantages : Mycoplasma & L-forms are resistant

D - Members

Penicillins	Cephalosporins	Carbapenem	Monobactam	Glycopeptides	Bacitracin	Cycloserine

E-Mechanisms of bacterial resistance

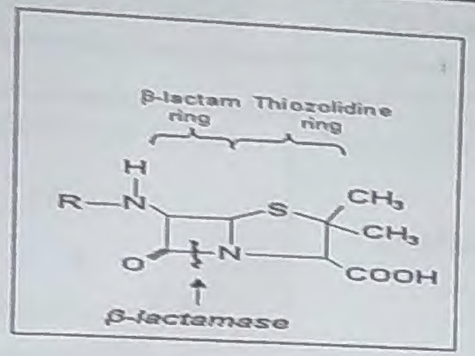
1-By destructive enzymes (coded by R plasmid)

β lactamase (penicillinase)

of Staph aureus

⋮ Destroy

Some penicillins & cephalosporins



ESBLs (extended spectrum β lactamase)

of G-ve bacilli

(arise by **mutation** in genes on plasmids coding for β lactamase)

⋮ destroy

As before + *monobactam (aztreonam) &carbapenem*

2- Alteration of transpeptidase

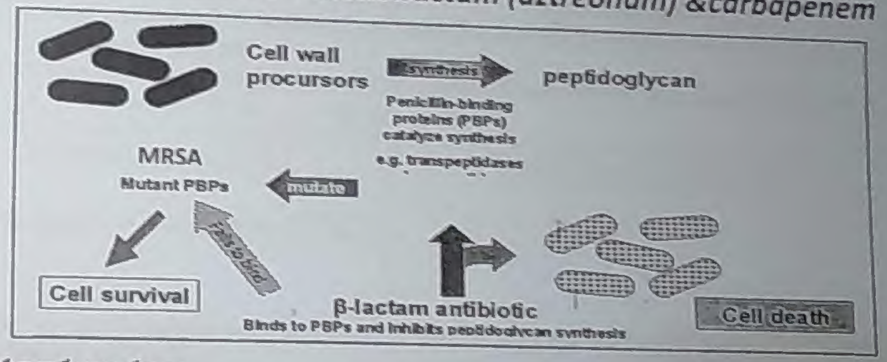
By **MRSA** (Methicillin resistant Staph.aureus)

⬇

Resistant to all β lactams,

including those resistant to β lactamases e.g

methicillin, cloxacillin & nafcillin



F- Important notes

	Advantages	Disadvantages
1-Penicillins		
i.PenicillinG , ampicillin , amoxicillin	Broad spectrum	Destroyed by β lactamase (penicillinase)
ii.Methicillin , cloxacillin , nafcillin	Not destroyed by β lactamase	Narrow spectrum: *G+ve only*
2-Glycopeptides Vancomycin & teicoplanin	Only TTT of MRSA	Narow spectrum: *G+ve only*

Cell wall deficient bacteria

Mycoplasma	Protoplasts	Spheroplasts	L-forms
A-Inducer			
Natural (no inducer)	*Lysozyme* on *G+ve* bacteria ↓ **Destruction** of PG	*Penicillin* on *G-ve* bacteria ↓ ⊖**synthesis** of new PG	*i.Penicillin on G+ve or –ve* bacteria ↓ ⊖ **synthesis** of new PG *ii.Spontaneous*
B-Structure & Characters			
L a c k r i g i d c e l l w a l l			
i.No defined shape ii.Resistance to antibiotics acting on CW	**Complete absence** of CW	**Remenants** of PG (damaged or weakened CW)	**Vary** in size &shape
C-Osmotic sensitivity			
NO (stable)	*Sensitive→ vary in size with OP of suspending medium*		More stable
D-Multiplication			
Yes	*No*	Yes	Yes
E-Reversion			
No		5	Yes, **if penicillin is removed** ↓ *Relapse of infection*

Mycoplasma

Natural absence of CW

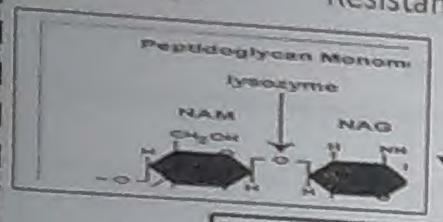
No shape

Resistance to antibiotics on CW

No reversion

Compensatory ↑ in CM thickness

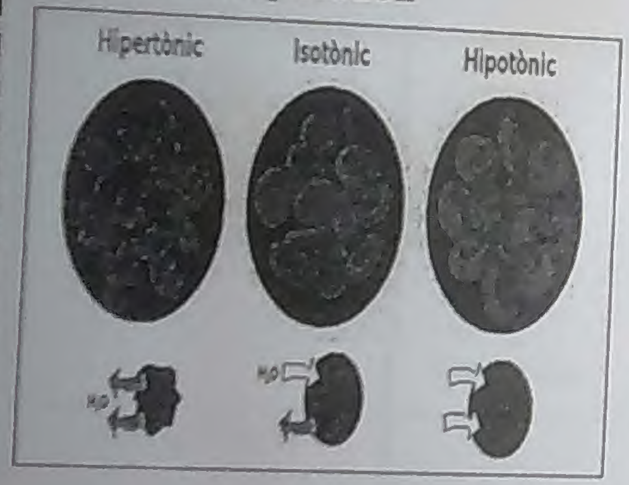
Osmotic stability ↔ Multiplication



Protoplast

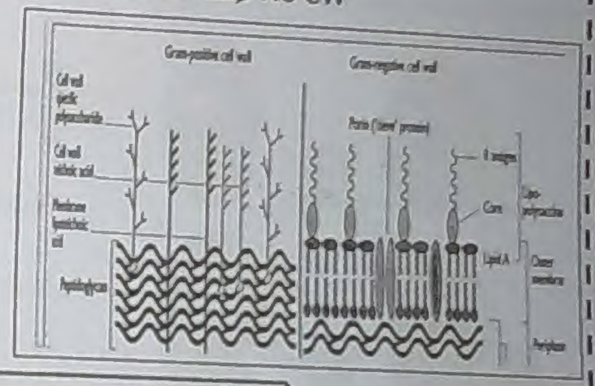
Lysosymes on G +ve in vitro → Destruction of PG & techoic acid → No CW

No multiplication



Osmotically sensitive

Isotonic M	Hypotonic	Hypertonic M
No rupture	Ballooning &rupture	Shrinkage



Spheroplasts

Penicillin on G-ve in vitro → ⊖ CL of PG

Loose (remenant) PG

Osmotically sensitive a.s. (protoplast)

Remaining CW layers are intact (OM)

Multiplication (if maintained in Isotonic M)

L (Lister institute) forms

Rarely Penicillin on G +ve or G –ve → partial ⊖ of CL of PG in few bacteria during infection

Part of PG remains intact & remaining CW layers are intact

Osmotically more stable

Survive (although the body is Hypotonic) & multiply (new L-forms) for sometime

Continuous use of Penicillin

Kill them

Early stoppage of penicillin

CW revert to original form → relapse

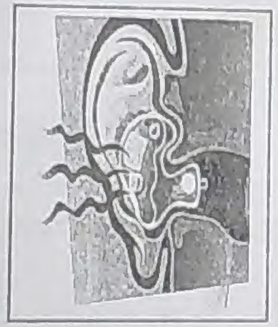
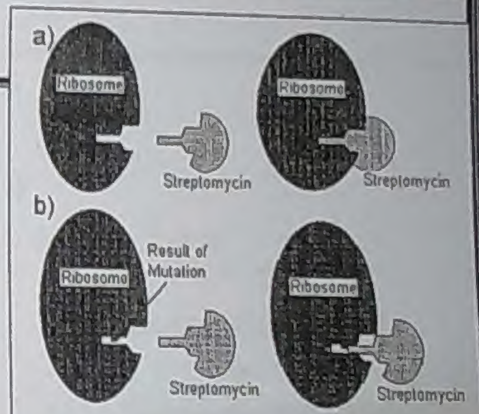
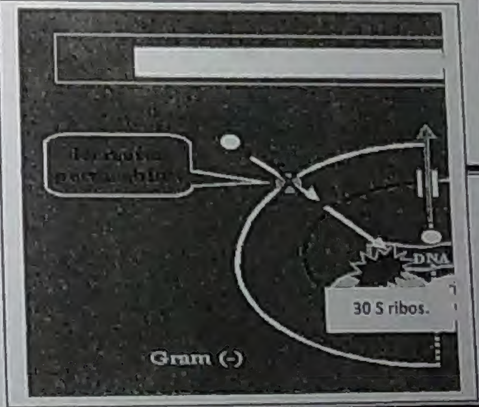
5'

Antibiotics inhibiting protein synthesis

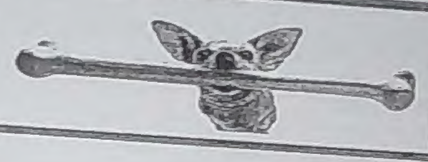
Advantages

Selective toxicity : Bacterial ribosomes are different from human ribosomes

A- Drugs acting on 30S ribosomes

	S.E.		Mechanisms of bacterial resistance	
1-Aminoglycosides	**Nephrotoxic**			
i.Streptomycin	Toxic to 8^{th} CN ⟶ **deafness**		*Change of streptomycin receptor (target R) on 30S ribosome* (due to spontaneous **mutation** of **chromosomal gene**)	
ii.Amikacin			*↓ permeability to the drug* Change in **OMP** ⟶ ⊖ of active transport of drug	
2-Tetracyclines	**Permanent staining of child teeth** (if given in pregnancy or early in life)		As amikacin	

B - Drugs acting on 50S ribosomes

	S.E.	Mechanisms of resistance
1-Chloramphenicol	BM depression 	
2-Macrolides (erythromycin)		*Destruction by acetyl transferase*
3-Azalides		
4-Clindamycin	Superinfection → pseudomembranous colitis	

Superinfection

Etiology

Prolonged use of BS antibiotic to treat pathogenic bacteria

↓

Suppression of sensitive bacterial flora

↓

Overgrowth of flora which are

Resistant *Potentially pathogenic*

Examples

Candida

↓

Oral thrush

or vaginitis

Clostridia difficile

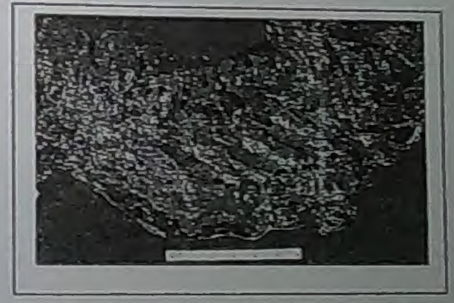
↓

Pseudomemb.colitis

(drug associated colitis)

↓

Diarrhea



Flora

- ✓ Organisms that grow in healthy persons in many sites e.g Skin ,GIT,vagina
- ✓ Some are non pathogenic & others are potentially pathogenic

Pathogenecity of flora

Low n + non Virulence → **Non** pathogenic

Low n + ***Low virulence*** → **Potentially** pathogenic

❖ **Flora are kept in low n by**

Immunity

↓ immunity → **Ds**

Mutual ⊖ **between different families**

Overgrowth of 1 family → **Ds**

Superinfection

BSA

Candida (PP) — Other bacterial flora in mouth

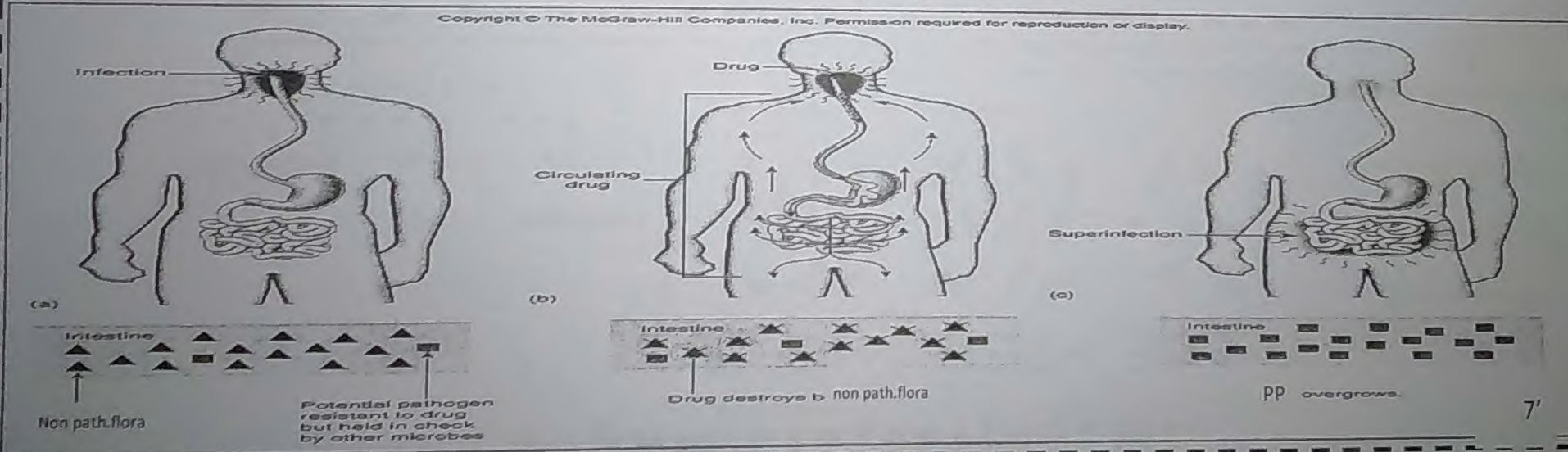
No more ⊖

Overgrowth → ***SI***

BSA : Clindamycin

Cl.difficile — Other anerobic flora in colon

Overgrowth → ***SI***



Complications of chemotherapy

1-Drug toxicity

Etiology

Overdosage or prolonged use especially in:

♫ Children & old people ♫ Pregnant ♀

Examples

- Polymixins (E)
- Aminoglycosides & Streptomycin(E)
- Tetracyclines (E)
- Chloramphenicol (E)

2-Allergy (Hypersensitivity)

Etiology

Drugs acting as **haptens**

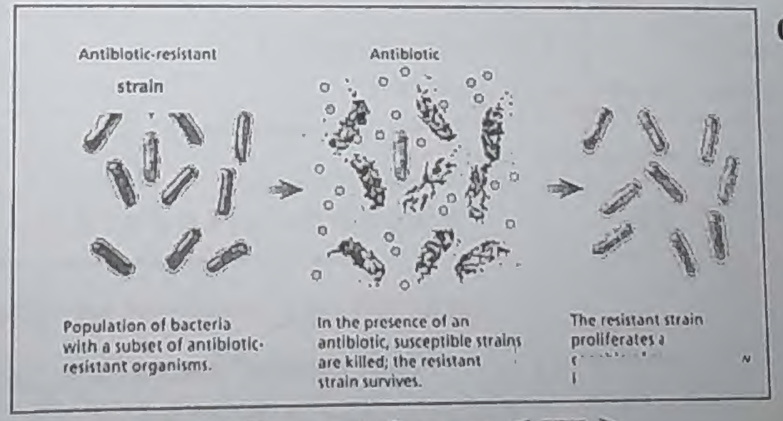
Examples

- Penicillins → Anaphylactic shock
- Sulfonamides (topical) → Contact dermatitis

3-Development of resistance

Etiology: misuse

- **Low** dose
- **Interrupted** course
- **Wrong** choice
- **No indication**



Consequences

Emergence & overgrowth of resistant org.

4-Superinfection(E)

Antibiotics inhibiting nucleic acid

Inhibition of DNA		Inhibition of RNA
Indirect inhibitors	*Direct inhibitors*	

I – Inhibition of DNA

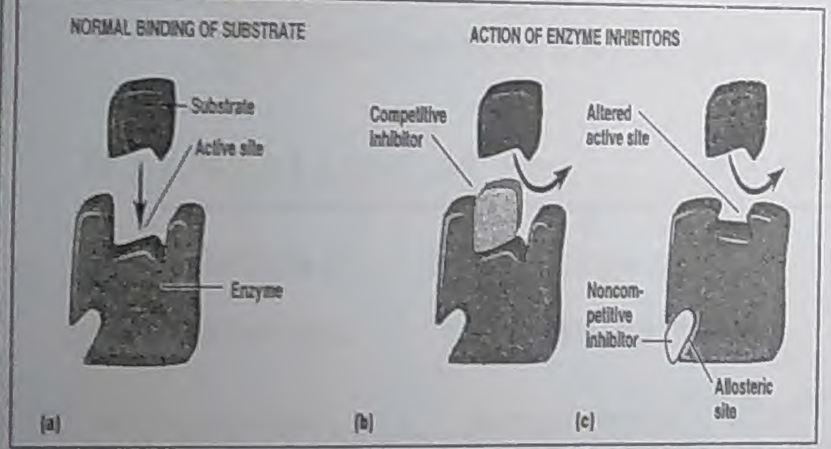
A - Indirect inhibitors

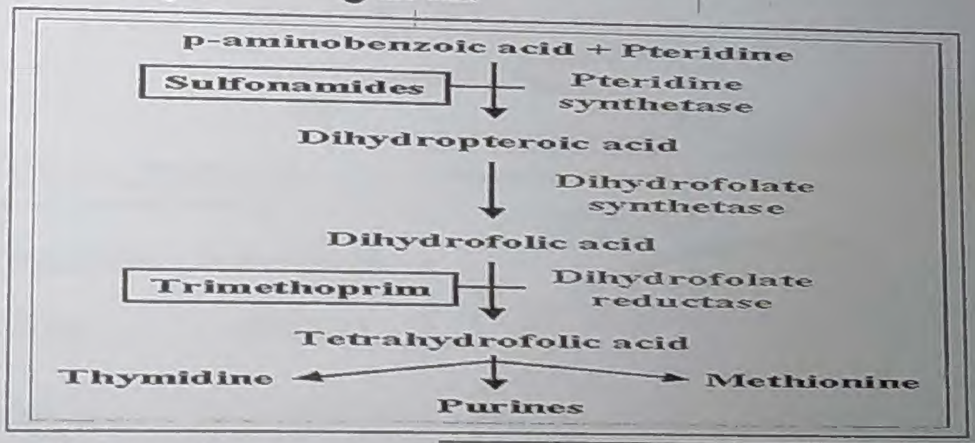
Act by **competitive inhibition**

PABA ⟷ **Sulfonamide** (*Enzyme*) → Folic acid → **Dihydrofolic acid** ⟷ **Trimethprim** (*DHFA reductase*) → TetraHFA → DNA

Structural analogues — ⊖ the enz.

Structural analogues — ⊖ the enz.

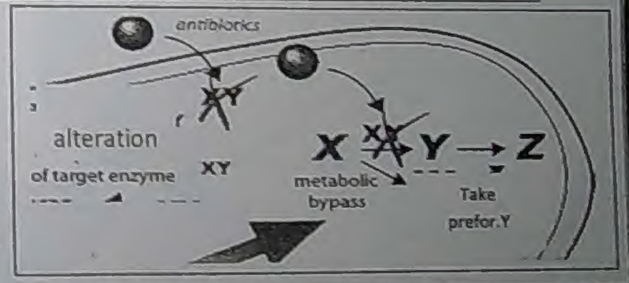




Resistance to sulfonamides : metabolic changes

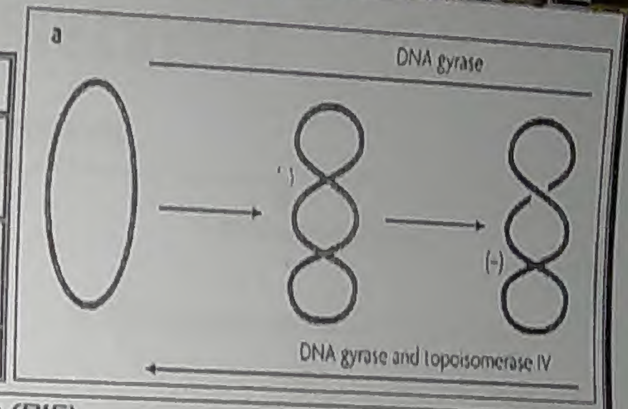
Production of altered enzyme having higher affinity for PABA

Bypassing the reaction ⊖ by the drug Using **preformed human F.A.** instead of synthesizing F.A. from human PABA



B - Direct inhibitors

	M.O.A	Examples	Uses
1-Quinolones	⊖ *DNA gyrase*	• Nalidixic acid • Ciprofloxacin	
2-Azoles : Nitroimidazoles	*Breaks DNA*	Metronidazole	i.Anaerobic bacteria ii.Protozoa
3-Novobiocin			

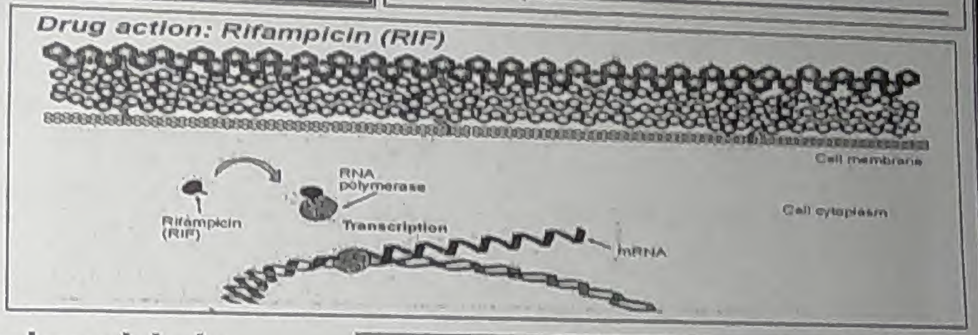


II-Inhibition of RNA

Rifampin

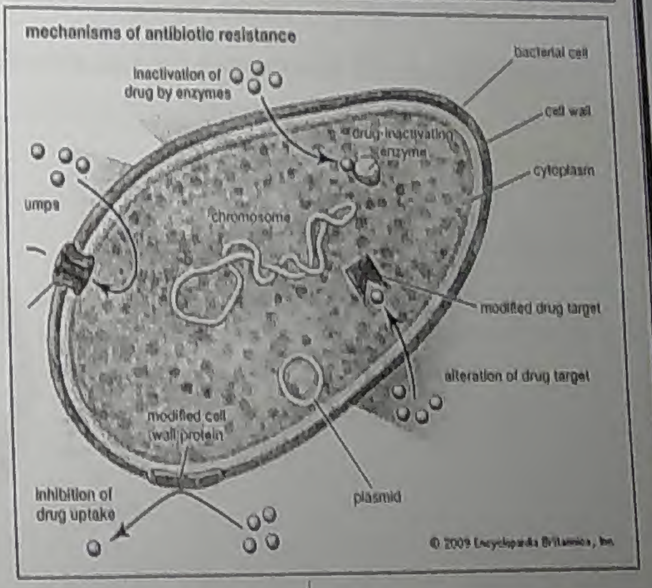
⊖ *RNA polymerase*

⊖ transcription of RNA from DNA



Mechanisms of res ...nce to antimicrobials

1-Production of destructive enz.	2-Change of target receptor	3 -↓ permeability to drug	4-Metabolic changes
♣ β lactamase & ESBLs (M)	Streptomycin (M)	Amikacin & Tetracycline (M)	Sulfonamides (M)
♣ Acetyl Transferase (M)			



Origin of ant tic resistance

A - Genetic origin

Extrachromosomal

Plasmid

e.g R plasmid coding for β lactamase

Destroy β lactam ring

of penicillins & cephalosporins

Spread by

- ♦ Conjugation
- ♦ Transformation
- ♦ Transduction

Complex

transposons

Chromosomal

Spontaneous mutation of a gene

Change in **target receptor**

e.g change of streptomycin receptor

on 30S ribosome

B - Non genetic (phenotypic) origin of resistance to antibiotics

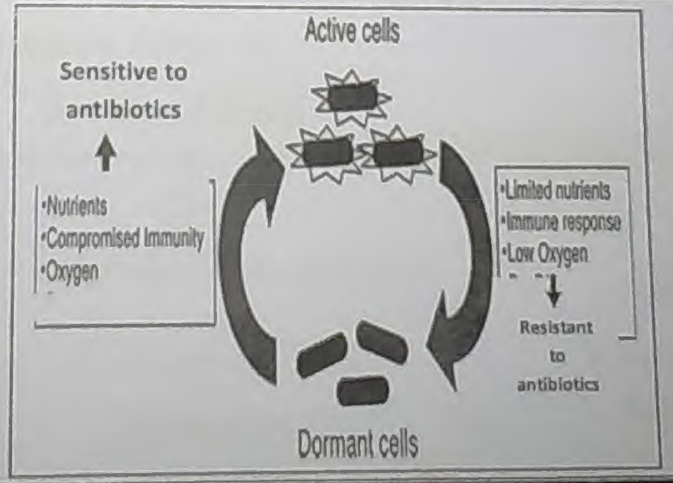
Metabolic inactivity

Dormant (metabolically inactive) TB

survive in tissues for many years &don't multiply

Are resistant to antibiotics which act ***only on***

metabolically active &replicating bacteria



Lack of target structure

Mycoplasma & L-forms

are resistant to antibiotics

that ⊖ CW

Antibiotics interfering with CM functions

A-Antibacterials: Polymixins B & E (colistin)

Mode of action

M.O.A.

Disrupt CM

Escape of macomol.&ions

Lysis

Advantages

Selective toxicity

CM of bacteria differs from CM of humans

More disrupted

Disadvantages

Highly toxic

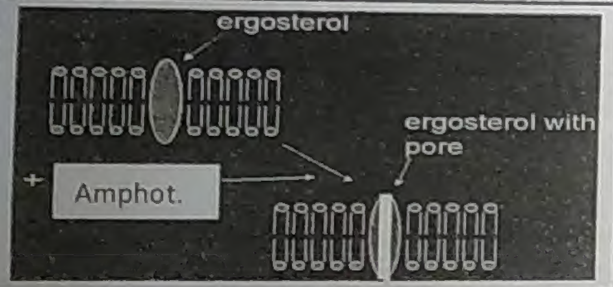
Used only topically

S.E.

- Nephrotoxic
- Neurotoxic

B - Antifungals

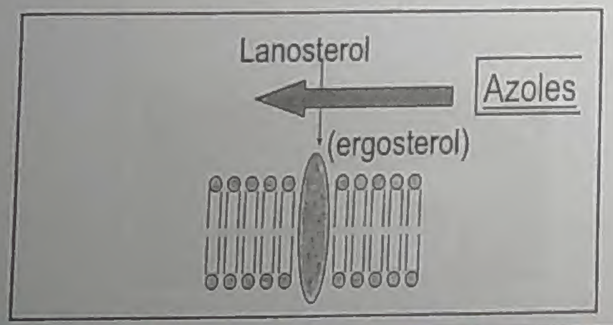
1 – Polyenes

Amphotrecin B	Nystatin
Bind **sterols** in CM → ↑ fluidity → **pore formation** → loss of ions & small mol. → Lysis **(cidal)**	
	Less toxic as it has ***greater affinity for ergosterol*** (in fungi) than cholesterol (in humans) Ttt of **systemic** fungal inf.
	More toxic Ttt of **local** fungal skin & **MM** inf.

2 – Azoles

M.O.A.

⊖ synthesis of ergosterol



Uses

***Broad spectrum** antifungal*

Ttt of Candida & Dermatophytes

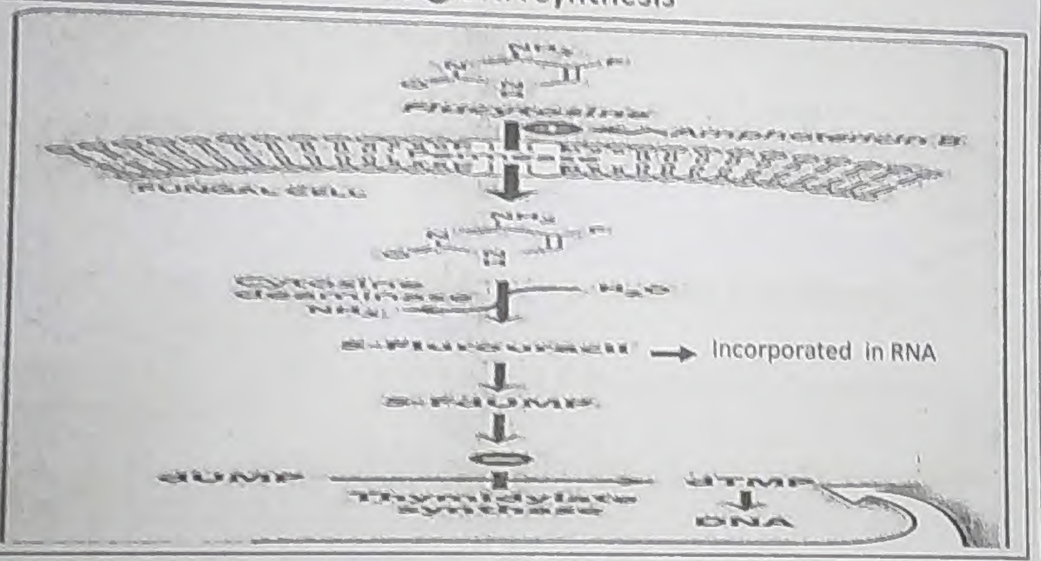
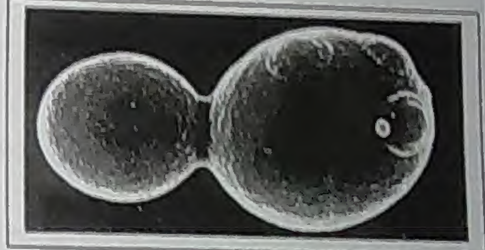
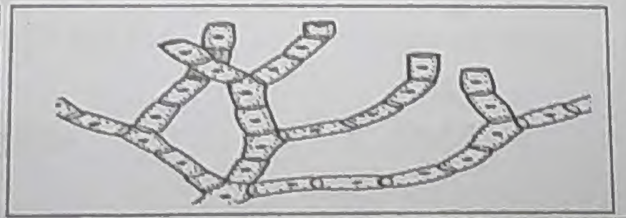
Examples

Systemic	Topical (toxic)
♦ Imidazole	♣ Miconazole
♦Triazole	♣ Clotrimazole

Antifungal drugs

A-Drugs acting on CM : poleynes & azoles

B – Other Antifungal drugs

	M.O.A	Uses
1-Flucytosine **(Static)**	i. ⊝ RNA synthesis Incorporated into fungal RNA (analogue to **Uracil**) ii. ⊝DNA synthesis 	Ttt of systemic fungal inf. by **Candida & Cryptococcus** (+ Amphotrecin)  
2-Griseofulvin	⬭ hyphal growth (concentrated in keratinized tissues)  	Ttt of **dermatophytes** inf. (in skin , hair & nails)

Antibiotic mbinations

A-Indications & applications

1-Severe undiagnosed infections

Septicaemia

2-Mixed infection

3-Prolonged course of ttt e.g ttt of TB

- Prevent resistance
- ↓ toxicity (↓ dose of each)

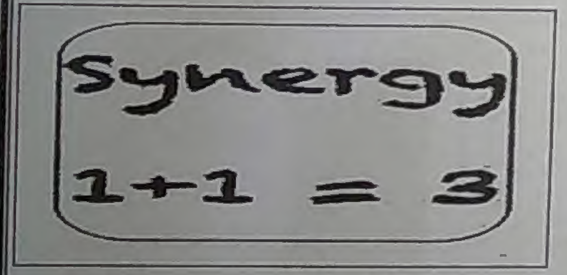
4-Complete eradication of org.

Avoid complications

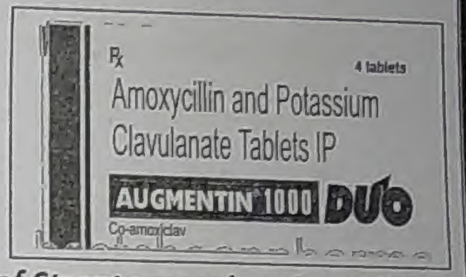
5-To obtain synergism

Definition

The combined effect of both drugs is > sum of them



Examples



Ttt of bacteria producing β lactamase

Clavulonic acid (β lactamase ⊖) *potentiates* action of amoxicillin (β lactam) → **Coamoxiclav**

Ttt of Streptococcal endocarditis

β lactam drug *potentiates* action of aminoglycosides

B-Effects

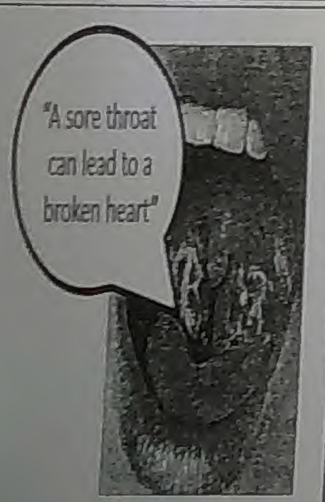
The combined effect of both drugs may be

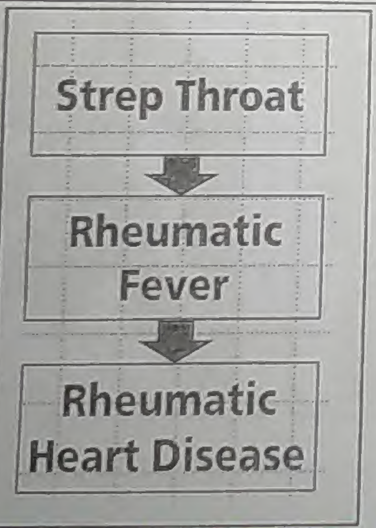
- < most potent of them → **Antagonism**
- = most potent of them → **Indifference**
- = sum of them → **Addition**
- > sum of them → **Synergism**

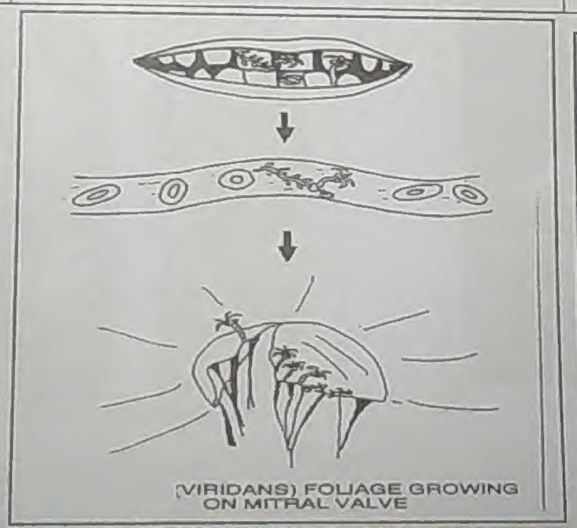
Prophylactic u f antimicrobials

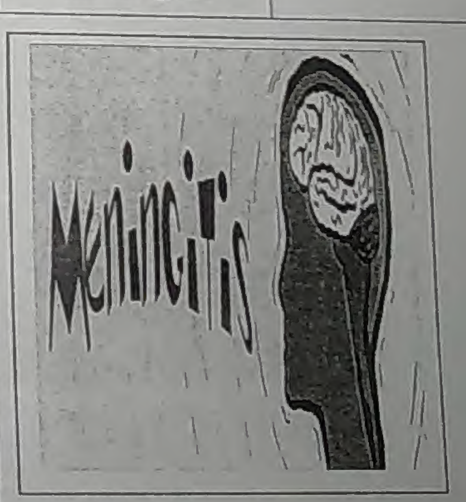
I-Medical prophylaxis

Rheumatic fever	Subacute bacterial endocarditis	Meningitis	Cholera
Penicillin G every 4 ws to pts with **rheumatic fever**	Single dose of **amoxicillin** before **dental operations** **or tonsillectomy**	**Rifampicin** for **close contacts of case**	Tetracycline
⬇ Prevent ***recurrence*** of throat inf. by ***Streptococcus pyogenes***	⬇ Prevent SABE (by Strep.viridans) in pts with ***congenital or rheumatic heart ds***	⬇ Prevent ***meningococcal meningitis***	⬇ Prevent ***cholera***









II-Surgical prophylaxis

Aim

Prevent spread of inf. to a ***clean surgical field***

Criteria

Indications

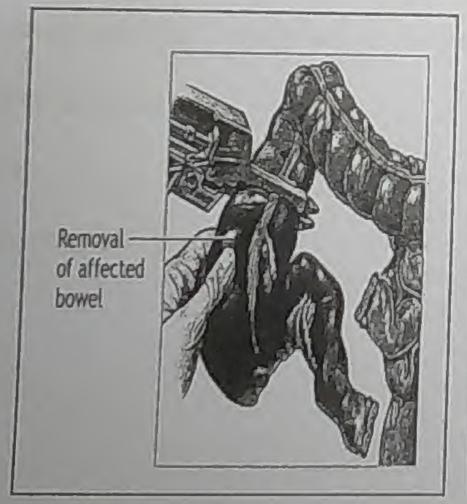
- ***High risk of infection*** e.g colon resection
- ***Severe consequences of infection*** e.g joint replacement
- ***High risk patients***

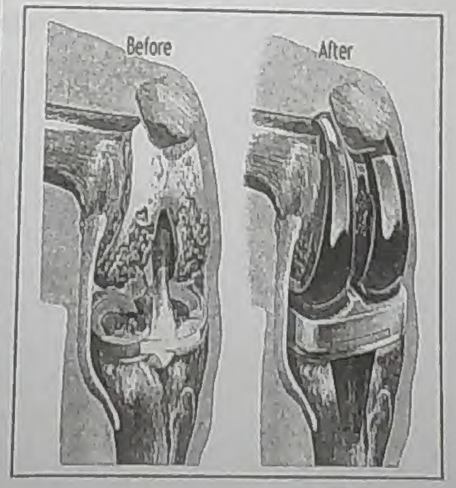
Time

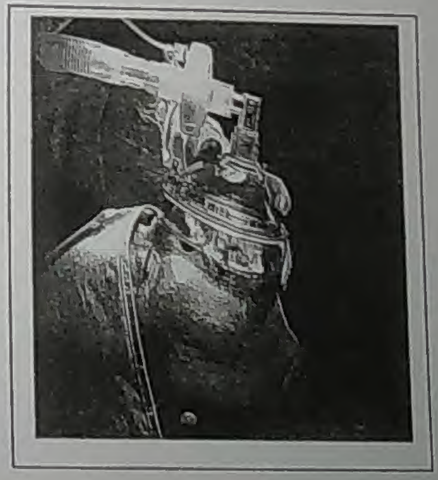
- **Before** induction of **anaeshesia**
- **No more** than 24 hrs after oper.

Selection

Active against org. encountered in operation







Essay questions

1- Give an account on Beta lactam antibiotics regarding mechanism of action and bacterial resistance.

2- Define MRSA,mention its mechanism of resistance &treatment.

3- Antibiotics acting on nucleic acids.

4- Antibiotics acting on ribosomes.

5- Mechanisms of antibiotic resistance.

6- Genetic origin of resistance.

7- Non genetic origin of resistance.

8- Antibiotic combinations as regards indications and applications in treatment (mention all the page).

9- Surgical prophylaxis by antibiotics.

10- **Give reason**: Prolonged antibiotic therapy should be avoided

(Due to drug toxicity , resistance & superinfection).

11- **Give an account on** :

a. Synergism.

b. Superinfection.